CLASSICS OF
CARDIOLOGY

Anatomic drawings of the heart by Leonardo da Vinci (1512)

CLASSICS OF CARDIOLOGY

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A Collection of Classic Works on the
Heart and Circulation with Comprehensive
Biographic Accounts of the Authors

Fifty-Two Contributions by Fifty-One Authors

BY

FREDRICK A. WILLIUS, M.D., M.S. in Med.

Former Chief, Section of Cardiology, Professor Emeritus of Medicine,
The Mayo Foundation for Medical Education and Research,
The Graduate School, The University of Minnesota

AND

THOMAS E. KEYS, A.B., M.A.

Librarian, The Mayo Clinic; Assistant Professor of the History
of Medicine, Mayo Foundation for Medical Education and Research,
The Graduate School, The University of Minnesota
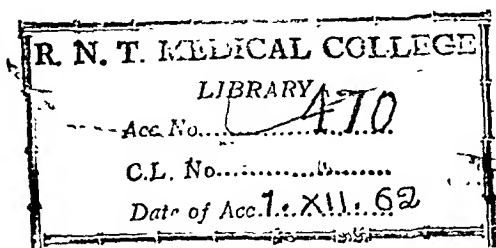
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CONTENTS

VOLUME ONE

VOLUME TWO

ILLUSTRATIONS

VOLUME ONE

VOLUME TWO

CLASSICS OF
CARDIOLOGY

1831

JAMES HOPE

DESCRIPTIONS OF CARDIAC ASTHMA, STENOSIS OF
THE PULMONARY VALVES, AND CARDIAC NEUROSIS

JAMES HOPE

Engraving from Memoir of the late James Hope, M.D., by Mrs. James Hope, published in 1848

(Courtesy Charles C Thomas.)
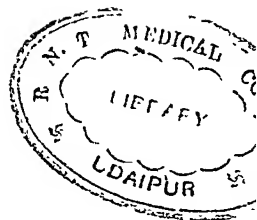
JAMES HOPE

(1801-1841)

"[He] attained great eminence, and large practice, at an age when most physicians are only beginning to be heard of. His success was not owing to the patronage of any great man, nor to any of those fortunate accidents which have occasionally brought physicians suddenly into notice. He was indebted simply to his own talents, his active humanity, the weight of moral character, and the force of industry, for his rapid elevation."

—Dr. Klein Grant, in Preface to

Memoir of the late James Hope.

*J*AMES HOPE was born in Stockport in the county of Cheshire, England, on February 23, 1801. His father was a successful merchant and manufacturer and desired his son, James, to become a merchant. The young man was educated at the Macclesfield Grammar School, and at the age of eighteen, he decided to become a lawyer. In the year 1819 occurred the Manchester riots, culminating in the so-called Battle of Peterloo on August 16, which so enraged the poet, Percy Bysshe Shelley, that in his "Mask of Anarchy, Written on the Occasion of the Murder at Manchester," he excoriated England's foreign secretary of the time, Robert Stewart Castlereagh, in the lines:

> "I met Murder on the way—
> He had a mask like Castlereagh."

In 1822, when Viscount Castlereagh cut his throat, Lord Byron wrote:

> "So He has cut his throat at last!—He! Who?
> The man who cut his country's long ago."

Young Hope enlisted in, and spent about a year with, the Yeomanry Lancers, a body of men raised to cope with the disturbance. When he subsequently returned home, his father suggested that he become a physician. Hope did not like this idea, but decided to give medicine a trial provided, after he became a physician, he should be allowed to practice in London.

In 1820, after a period spent at Oxford, Hope began the study of medicine at Edinburgh. The next year he was asked by Dr. James Bardesley of Manchester to join the Royal Medical Society of Edinburgh. At a meeting of this society he presented a paper on the heart, and it was so well received that he decided to write a work on diseases of the heart.

In 1824, Hope was elected house physician to the Edinburgh Royal Infirmary, and in 1825 became house surgeon to this institution. Later, that same year, he received the degree of Doctor of Medicine from the University of Edinburgh. He chose as his dissertation subject "Aneurism of the Aorta." In his thesis (1825) he proved that it was possible to diagnose aneurysms of the aorta during the life of the patient, which, according to Laënnec, was not possible.

Hope, feeling that it would better qualify him for the practice of medicine, went to London to study surgery at St. Bartholomew's Hospital, in 1826. In the spring of

A TREATISE ON THE DISEASES OF THE HEART AND GREAT VESSELS*

By

J. HOPE, M.D., F.R.S.

Of St. George's Hospital; formerly Senior Physician to the St. Marylebone Infirmary; Extraordinary Member, and formerly President, of the Royal Medical Society of Edinburgh, etc.

CARDIAC ASTHMA†

AMONGST the diseases of the heart may be justly reckoned one of the forms of the malady termed in common language *asthma*. This has been too much regarded as independent of disease of the heart. Long treatises have even been written upon it without ever mentioning disease of this organ as one of its causes. It is, therefore, necessary to notice the subject formally in this place, not only for the purpose of showing the magnitude of the error, but of making the reader acquainted with all the habitudes and aspects of a complaint, which is perhaps the most distressing in the whole catalogue of human maladies.

It is established by the concurrent testimony of all moderns conversant with diseases of the heart, that these diseases, no less than those of the lungs, may constitute the organic causes of asthma.

A theoretical consideration of the subject leads, in my opinion, to the same conclusion; for, on tracing asthma back to its source, we shall find that, whatever be its proximate cause in different cases, it is connected, in all, with the same ultimate circumstance; namely, inadequate oxygenization of the blood, and the resulting want of breath, which, through the "incident excito-motory" branches of the pneumogastric, excites the "reflex" action of the "true spinal" nerves on the muscles of respiration. For instance, inadequate oxygenization of the blood results in all ordinary cases from one or more of three proximate causes: viz.

A. Insufficient admission of air into the bronchial tubes and air-vesicles.

B. Insufficient exposure of the blood to the air admitted, in consequence of a less pervious state of the mucous membrane than natural.

C. Insufficient admission of blood into the lungs.

It will be found that, to one or more of these causes, all the varieties of dyspnoea and asthma are referable.

.   .   .   .

4. *Diseases of the Heart.*—Sometimes, from this cause, blood exists in the lungs in excess; as is the case when the right ventricle is hypertrophous, or the left side of the heart obstructed; or, still more, when these two affections co-exist; also when the circulation is merely accelerated, as by palpitation, running, or by slighter efforts in corpulent persons. Now, under all these circumstances, there is inadequate oxygenization of the blood; or, in other words, there is an excess of venous blood in the lungs; first, because the quantity of blood admitted exceeds its due proportion to the air in the organ; secondly, because the engorgement of the mucous membrane on which the blood ramifies, constricts the bronchial passages, and prevents the free ingress of air, as proved by the feebleness of the respiratory murmur. Hence, want of breath is a necessary consequence of an excess of blood in the lungs.

Sometimes blood does not enter the lungs in sufficient quantity, constituting the third cause of inadequate oxygenization; and this may arise from the weakness of the right ventricle, from an obstruction in its mouth, or from increased resistance on the part of the lungs; as, for instance, during sleep, when the respirative function is less active. Hence results the stimulus of want of breath, and dyspnœa. Cases exemplifying this will shortly be adduced: meanwhile it may be illustrated by a simple physiological experiment, viz. by making and sustaining a full *expiration*. This is attended, not only with a deficiency of air, but also with a deficient influx of blood into the lungs, as is proved by the lividity of the face which ensues, by the elevation of the fontanel in infants; by the rise of blood in a tube inserted into the jugular vein; and lastly, by experiment; for I have demonstrated above, . . . that, on suspending artificial respiration in a rabbit, the heart *instantly* became gorged, of a black colour, and distended to nearly double its natural size—a phenomenon which renders it sufficiently manifest that, when the lungs are exhausted of air, the blood does not freely enter them. Now, the sensation of want of breath experienced on making a full expiration is familiar to every one, and it becomes intolerable if the expiration be long sustained.

5. *Spasmodic constriction of the bronchial tubes* is presumed to exist, first, because, according to the researches of Reisseissen and others, the bronchial tubes are provided with muscular fibres, and all muscles are liable to spasm: secondly, because asthma is occasionally found to occur without any organic cause (so far at least, as our senses enable us to judge) sufficient to account for it: thirdly, because every form of organic disease above described, both of the lungs and the heart, may exist without causing dyspnœa of such intensity and of such a character as to constitute *asthma* properly so called. Thus, many have intense chronic bronchitis and profuse expectoration without any asthmatic dyspnœa; and I have known a patient with a contraction of the mitral orifice to the size of a small pea, and likewise with dilatation and softening of the heart and profuse expectoration,

pass through a period of ten years to her grave without ever experiencing a paroxysm of asthma, though a few steps across the room were sufficient to excite dyspnœa. (Mrs.—l—n.)

Hence, I apprehend that whatever be the organic cause of asthma, it requires for its production the superaddition of a state of the nervous system leading to spasmodic constriction of the bronchial tubes. Why some should exhibit this state and others not, is one of the arcana of the nervous system; but observation has shown that the state is constitutional and often hereditary.

Admitting that the spasmodic constriction of the bronchial tubes does take place, it is obvious that it will more or less close these tubes against the ingress of air; and this closure, again, by preventing the free expansion of the lungs, will impede the influx of blood. Whence there is a double cause for the inadequate oxygenization of the blood, and consequently, for the production and maintenance of the asthmatic paroxysm.

From all that has been said, we are now led to the resulting inquiry— what is the essential difference between asthma from disease of the heart and that from disease of the lungs. Putting aside that variety of asthma which, as not being attended with any *visible* organic derangement, (though it is, notwithstanding, highly probable that one exists,) may be regarded as mainly, if not wholly spasmodic, there does not appear to be any essential difference between the remaining varieties. Their organic causes are diversified, but they all ultimately produce the same effect, and it is the effect which constitutes the essence of the disease. This effect is inadequate oxygenization of the blood, which causes "excitant" want of breath; and this, when the case is really asthmatic, i.e. more than what may be called mere dyspnœa, occasions spasmodic constriction of the bronchial tubes, and its consequence, the asthmatic paroxysm.

We now proceed to the more particular consideration of asthma from disease of the heart.

This variety comprises, according to my observation, by far the greater proportion of the most severe and fatal cases of the disease. Some are of the opinion that in other varieties the patient experiences an equal degree of suffering during the continuance of the paroxysm. I cannot say that this is consistent with my own observation. Though the same words may suit for the delineation of an attack of each variety, my feeling and conviction is, that I have never seen the patient suffer such intense and suffocative agony as in the variety from organic disease of the heart.

Until the discovery of auscultation had in some degree dissipated the deep obscurity of the affections of this organ, the fact that they were a cause of asthma was scarcely known: and, even at the present day, there are few errors more common than that of attributing asthma to other causes, when it originates solely in the heart. For instance, a theory of this description which has for the last half century been more widely disseminated than

perhaps any other, consists in ascribing asthma to a spasmodic or convulsive contraction of the external muscles of respiration, much dependent on habit.

Now, the action of these muscles, so far from being morbid or dependent on habit, is a natural instinctive and salutary effort to prevent suffocation, the stimulus to which consists in an exaggeration of that which excites the muscles in ordinary respiration—namely, as above explained, the want of breath, resulting from inadequate oxygenization of the blood. Nothing is more common, for instance, than to see a patient with diseased heart, while sleeping tranquilly, start up and begin to respire with violence. Here it is obvious that the necessity for violent respiration preceded the act; and the necessity depends on impeded transmission of blood through the heart and lungs; for starting is invariably accompanied by palpitation, and preceded by frightful dreams, or some sensation of praecordial distress, indicating an obstructed circulation. I have frequently examined the heart and lungs by auscultation immediately before the supervention of a paroxysm of dyspnoea, and have always found that the heart began either to palpitate, or to act in that irregular, confused, and, as it were, struggling manner, which denotes its engorgement. I was therefore enabled to tell the patient that difficulty of breathing was coming on, to which with some astonishment, he would reply in the affirmative, being himself forewarned of the approaching accession by a feeling of anxiety and straitness in the praecordia. The fact is so universally true, that any one may satisfy himself of it by entering an hospital and gently placing a patient with orthopnoea from disease of the heart, in a rather uneasy position, when the series of phenomena described will become manifest.

Dr. Burrows communicated to me the particulars of a case, recently under his observation, in which the respiration was alternately violent and tranquil under the following circumstances. The patient dozed for a few minutes at a time, during which his complexion became livid, and his pulse more and more feeble, oppressed and irregular. He then started up, and, after a few violent wheezing respirations, relapsed into the same calm doze. In this case the mitral orifice was contracted to the size of a pea. Now, there can be little doubt that as, during sleep, the stimulus of want of breath is less felt, and the muscles of respiration are, consequently, less excited by it,—in simple language, as the respiration is more feeble during sleep, the lungs were not, in the present case, kept sufficiently expanded to admit of an adequate circulation through them: whence ensued engorgement of the heart and venous system of the body, with insufficient arterialization of blood in the lungs, and the necessity for breathing resulting from it, which series of phenomena was relieved by the succeeding violent respirations. I have frequently observed this series of phenomena in a greater or less degree: occasionally even in coma. In another case, violent gasping and wheezing respiration, lasting from a few seconds to two or three minutes,

occurred at intervals of four or five minutes, during which the patient dozed, even though sitting erect on a stool and undergoing a stethoscopic examination; and this series of actions continued so long as the patient remained disposed to sleep in that situation. In another case of great dilatation and softening, the precise symptoms described by Dr. Burrows occurred for the last week of the patient's life, whether he was awake or asleep, except when calmer sleep was procured by mild opiates. In a third case, a lady had, for several years, observed her husband's respiration while he was in the horizontal position, but not in the raised position, to be as follows:—after every four or five respirations calmly performed, succeeded a pause of a few seconds; then he started with a "convulsive motion of all his limbs, and a heaving of the shoulders." She had watched this continue for hours together, but he was unconscious of it, and generally slept soundly without frightful dreams. His disease was slight hypertrophy and disease of the aorta.

In all these cases, it is manifest that the action of the muscles of respiration was consecutive to the obstruction of the circulation, and that it was not dependent on any spasm of those muscles, but simply on the necessity for breathing, which instinctively excited them to a salutary preservative effort.

Asthma from disease of the heart often imitates the characters of the other varieties; and this perhaps for a very simple reason, that the lungs are in much the same state as in those varieties. Thus, it is *humid* or *humoral,* when there is permanent engorgement of the lungs, causing copious sero-mucous effusion into the air-passages, as in cases of contraction of the mitral valve. It is *dry,* when the engorgement is only temporary, as in cases of pure hypertrophy. It is *continued,* when there is a permanent obstruction to the circulation; and any of the varieties may be *convulsive,* when the heart has sufficient power to palpitate violently. The worst cases of convulsive asthma from disease of the heart are those of hypertrophy with dilatation and a valvular or aortic obstruction.

We shall now examine the state of a patient labouring under severe asthma from disease of the heart, and then take a more strictly medical view of the nature and progress of the asthmatic paroxysm.

The respiration, always short, becomes hurried and laborious on the slightest exertion or mental emotion. The effort of ascending a staircase is peculiarly distressing. The patient stops abruptly, grasps at the first object that presents itself, and fixing the upper extremities in order to afford a fulcrum for the muscles of respiration, gasps with an aspect of extreme distress.

Incapable of lying down, he is seen for weeks, and even for months together, either reclining in the semi-erect posture supported by pillows, or sitting with the trunk bent forwards and the elbows or fore-arms resting on the drawn-up knees. The latter position he assumes when attacked by

a paroxysm of dyspnoea—sometimes, however, extending the arms against the bed on either side, to afford a firmer fulcrum for the muscles of respiration. With eyes widely expanded and starting, eye-brows raised, nostrils dilated, a ghastly and haggard countenance, and the head thrown back at every inspiration, he casts round a hurried, distracted look of horror, of anguish, and of supplication: now imploring, in plaintive moans, or quick, broken accents, and half-stifled voice, the assistance already often lavished in vain; now upbraiding the impotency of medicine; and now, in an agony of despair, drooping his head on his chest, and muttering a fervent invocation for death to put a period to his sufferings. For a few hours—perhaps only for a few minutes—he tastes an interval of delicious respite, which cheers him with the hope that the worst is over, and that his recovery is at hand. Soon that hope vanishes. From a slumber fraught with the horrors of a hideous dream, he starts up with a wild exclamation that "it is returning." At length, after reiterated recurrences of the same attacks, the muscles of respiration, subdued by efforts of which the instinct of self-preservation alone renders them capable, participate in the general exhaustion, and refuse to perform their function. The patient gasps, sinks, and expires.

Such are the sufferings, in their worst form, of an asthmatic from disease of the heart. We have now to take a more strictly medical view of the nature and progress of the asthmatic paroxysm.

If about to be severe, it is generally preceded by certain premonitory symptoms, which, though not so marked as in ordinary asthma, are much of the same nature—probably because derangement of the circulation and imperfect oxygenization of the blood are present in both. In cardiac asthma, however, many of the nervous symptoms which characterise the ordinary varieties are often deficient. One of the most common and efficient exciting causes of cardiac, as of all other asthmas, is derangement of the stomach, the irritation of which extends to the heart, and stimulates it to inordinate action. The irritation, according to the theory of Sir Charles Bell, or the lately revived excito-motory views of Prochasca, is propagated through the medium of the par vagum, by which nerves the stomach and heart are closely associated. Accordingly, after a feeling of acidity, flatulence, or a load on the stomach from undigested food, often accompanied with abdominal distention, the patient experiences pain, weight, and constriction in the forehead and over the eyes, accompanied (if the case be one of hypertrophy of the left ventricle) with throbbing of the temples and the sound of rushing waters. He feels a sensation, scarcely to be defined, of oppression, and tightness and anxiety about the praecordia, frequently with slight palpitation. Sometimes the patient is drowsy, listless, restless, irritable, and impatient, not only of society, but of the attentions of friends: these symptoms, however, are, in general, more prevalent in ordinary asthma. The signs described afford the experienced asthmatic well-known assurance of the approaching attack.

They gradually become worse and worse, especially after a meal, and eventually burst into a paroxysm. The time of the accession is less regular than in ordinary asthma, being more dependent on the state of the heart, which is liable to accidental excitement at any moment, from a variety of causes. The fit, however, as in ordinary asthma, is, on the whole, more apt to supervene during the evening or early part of the night; and this, as appears to me, for two reasons: 1st. The recumbent position is unfavourable to respiration, the diaphragm being pressed upwards by the abdominal viscera, and the expansion of the chest being opposed by its own weight. 2d. During sleep, respiration is not assisted by the will, which, during the wakeful state, from the sensation of want of breath being more acutely felt, is ever ready to maintain the body in the position most favourable to breathing. From the co-operation of these two causes, therefore, the circulation becomes so far embarrassed before the patient is aroused to a sense of his condition, that it can only be relieved by those violent efforts which constitute the asthmatic paroxysm. He accordingly awakes, generally with a start, in a fit of dyspnoea, accompanied either with violent palpitation, or a distressing sense of anxiety in the praecordia and great constriction of the chest, as if it were tightly bound. He is compelled to assume a more erect posture, and intensely desires fresh, cool air; the respiration is wheezing, and performed with violent efforts of all the muscles of respiration, both ordinary and auxiliary. The inspirations are high and accompanied with apparently little descent of the diaphragm, and the expirations are short and imperfect. The surface is chilly, the extremities are cold, and the face is pale and sometimes livid.

In cases in which the pulmonary congestion is only *temporary,* as in hypertrophy either simple or with dilatation, there is no cough beyond a few slight and ineffectual efforts, producing little or no expectoration; and in such cases the fit subsides as soon as the engorgement of the heart and great vessels is relieved, which nature generally effects in two or three hours or less, by determining the blood to the surface and creating diaphoresis. In some instances, I have known this to be regularly accompanied with a copious secretion of pale urine and a purging alvine evacuation (case of May). In this case, the attacks recurred, according to the assertion of the patient, every night for several years.

The pulse, however full, strong and bounding at first, may, during the worst of the paroxysm, become feeble and small, and the sound and impulse of the heart may be diminished; and this, in cases even of hypertrophy; for the organ, being gorged to excess, is incapable of adequately contracting on its contents.

Such is the nature of an asthmatic fit when the pulmonary congestion is only temporary: the case is different when it is *permanent,* as in valvular disease and in some extreme cases of dilatation. For then, there is violent cough in suffocative paroxysms, accompanied, at first, with difficult and

scanty expectoration of viscid mucous, but ending gradually in a copious and free discharge of thin, transparent, frothy fluid, occasionally intermixed with blood. This evacuation, by disgorging the pulmonary capillaries, affords great relief to the cough and dyspnoea. As, however, the transudation of the matter to be expectorated into the air-passages, and its final elimination, are slow processes, paroxysms of this description are much more protracted than those of dry asthma from hypertrophy. They frequently last five or six hours, and I have known them to persist, with occasional remissions, for two, three, or more days. During the attack, the pulse is quick, small, and weak, often irregular and intermittent. The slowness which the latter characters sometimes appear to give it, has led some authors to suppose that the circulation through the heart is little disturbed in asthma. This is in some degree true in reference to other varieties of asthma; but it is always incorrect in reference to that from disease of the heart.

As the paroxysm subsides, the anxiety and constriction decrease, the respiration becomes less frequent, high, and laborious, and the pulse becomes slower, fuller, and more regular. But some degree of wheezing and tightness of the chest remain, and the paroxysm is very apt to return for two or three nights successively, and sometimes for a much longer period, until the lungs are freely unloaded by copious expectoration. It may, indeed, continue to recur at brief intervals for an indefinite period, or the patient may never be wholly exempt from some degree of asthmatic dyspnoea.

A severe asthmatic attack from disease of the heart is in general far more injurious in its consequence than one from an affection of the lungs.

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PART III, CHAPTER IX

SECTION IV

SIGNS OF DISEASE OF THE PULMONIC VALVES*

The signs of contraction of the pulmonic valves are the same as those of the aortic, . . . with this difference; that, from the vessel being nearer the surface the murmur with the first sound seems *closer* to the ear, and is on a higher key, ranging from the sound of a whispered *r* towards that of *s*. I have, however, known it fall below *r* when the circulation was feeble and slow, and the obstruction slight. It may be known that the murmur is not seated in the aorta, by its being inaudible, or comparatively feeble, two inches up that vessel; whereas, at a corresponding height up the pulmonary artery, it is distinct; also, by its being louder down the tract of the right ventricle than down that of the left (Bowden). It may be known that the murmur does not proceed from regurgitation through the auricular valves,

*Ibid. pp. 368-369.

by its being distinct along the course of the pulmonary artery, where auricular murmurs are either wholly inaudible, or very feeble and remote.

When a murmur in the pulmonary artery is considerably louder between the second, and third left ribs, close to the sternum, than opposite to the valves, and is there attended with impulse and purring tremor, dilatation of the pulmonary artery may be suspected (see *Dilatation of Pulmonary Artery*). In one instance I have known a murmur to be produced by complete ossification of the pulmonary artery penetrating deeply into the lungs (case of Lady R.).

When there is regurgitation through the pulmonic valves, a murmur accompanies the second sound. Its nature and diagnosis are the same, (the necessary inversions being made,) as in the case of aortic regurgitation, ... except that the pulse is not jerking (case of Rogers. A tremor attended).

I presume that purring tremor with the first sound may be occasioned by contraction of the pulmonic orifice, though I have not met with an instance verified after death: but I have met with three in which the tremor attended dilatation of the pulmonary artery (Weatherly, Bowden, and Miss L. P—r). A purring tremor occasioned by the pulmonic valves would be more readily felt than one occasioned by the aortic valves, because it would probably be transmitted as far as the space between the second and third ribs, (where it is out of the cover of the sternum,) provided the patient lay in the horizontal position, and inclined to the left side.

Disease of the pulmonic valves is so rare, that it ought never to be suspected unless the signs described are perfectly well marked, or unless there be patescence of the foramen ovale, or some other communication between the two sides of the heart,—states which experience has proved to be generally accompanied with contraction of the orifice in question.

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PART IV, CHAPTER II

PALPITATION FROM INORGANIC CAUSES, USUALLY CALLED NERVOUS, AND IMITATING DISEASE OF THE HEART*

There are few affections which excite more alarm and anxiety in the mind of the patient than this. He fancies himself doomed to become a martyr to organic disease of the heart, of the horrors of which he has an exaggerated idea; and it is the more difficult to divest him of this impression, because the nervous state which gives rise to his complaint, imparts a fanciful, gloomy and desponding tone to his imagination. Members of the medical profession are more apt than others to give way to these feelings; partly from their apprehensions being more keen, and partly from an impression too widely prevalent, that there is difficulty in distinguishing inorganic from organic palpitation, and, consequently, that they must remain in a

*Ibid. pp. 468-470.

state of anxious uncertainty. It may be said, for the consolation of such, that the diagnosis presents no difficulty to one who, to general signs, adds a knowledge of these afforded by auscultation and percussion. I repeat this opinion with increased confidence in the present edition, not only on the grounds of additional experience, but because the signs both of organic and inorganic disease will now be found much more precise and simple, in consequence of the new lights thrown on particular valvular diagnosis and on inorganic murmurs.

Inorganic palpitation presents certain varieties, which it is of the greatest practical importance to distinguish, as the treatment is different, and even opposite. It may be premised that, in all the varieties, the palpitation will, caeteris paribus, be greater in proportion as the patient is constitutionally of a more nervous, irritable temperament.

1. Palpitation dependent on dyspepsia, hypochondriasis, hysteria, latent gout, mental perturbations either of the exciting or depressing kind, excessive study with deficient sleep, and venereal excesses, constitutes the first variety, and forms a large class. When from these causes, it presents various degrees and characters. The slightest degree of it I should describe, from having occasionally experienced it, to be a tumbling or rolling motion of the heart, with a momentary feeling of tightness and oppression. It is referable to an intermission of the heart's action. In a further degree, as Abercrombie has well described, there is a series of quick, weak, fluttering, irregular beats, with slight anxiety, acceleration of the respiration, and a quivering sensation in the epigastrium: this may last from a few minutes to half an hour or an hour, and occur only at distant and irregular intervals, or repeatedly during the day, especially when the patient is startled. The next degree amounts to a perfect fit of palpitation, consisting in increased impulse, sound and frequency of the beats, sometimes with irregularity, and generally with more or less anxiety, dyspnoea, and even orthopnoea. The attack may be only occasional, or may occur several times a day, or may even last with little intermission for several days together.

The palpitation in question may be distinguished from that of disease of the heart, by the palpitation occurring only occasionally: by its not being excited, but, on the contrary, relieved by corporeal exercise of such a nature as would certainly disturb the action of a diseased heart: by its disposition to supervene while the patient is at rest, especially at the commencement of the night, when he lies wakeful in bed; by a fluttering in the epigastrium; by the general prevalence of nervous symptoms; by the affection being aggravated when the nervous symptoms undergo an exacerbation; by the pulse and the action of the heart being natural during the intervals between the attacks; and by the absence of valvular and aortic murmurs, and of undue impulse; "the shock, even when it at first appears strong, having little real impulsive force; for it does not sensibly elevate the head of the observer." (Laënnec.)

To this category some would add, an increase of the palpitation after meals, or when the stomach is deranged, and amelioration produced by dyspeptic remedies; but, as the stomach produces the same effects when there is disease of the heart, these signs are not pathognomic of nervous palpitation. To this point I would particularly direct the attention of practitioners; because many, in forming their diagnosis of the affections in question, regard the dyspeptic signs as paramount in value to all others, and are apt to refer to the stomach the palpitation which really belongs to organic disease of the heart.

Though the present variety of palpitation is often attended with various familiar nervous affections of the head, as pain or sensations of heat or of cold confined to particular parts and coming and going suddenly, temporary vertigo, tinnitus, and confusion of the sight; not increased by lying or stooping; it is not, when purely nervous and the patient not plethoric, accompanied with genuine signs of cerebral determination or congestion: there is no universal, throbbing headache with weight and tension, increased by stooping or the recumbent position: no stunning sounds and pains in the head on suddenly lying down or rising up: no permanent somnolency, apoplectic stupor, or regular apoplectic fits, as in hypertrophy, etc.

When it has been ascertained that the palpitation in question is independent of organic disease, the treatment presents no unusual difficulty, and is to be adapted to the nature of the exciting causes specified at the head of this division. It would be foreign to the subject of this work to dwell upon the particular remedial measures.
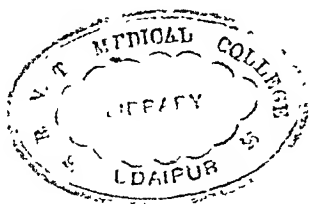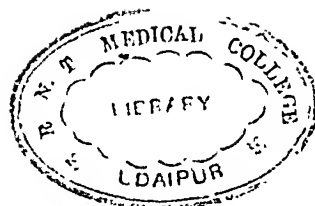
1832

SIR DOMINIC JOHN CORRIGAN

DESCRIPTION OF THE PULSE IN AORTIC
INSUFFICIENCY (CORRIGAN PULSE)

SIR DOMINIC JOHN CORRIGAN

SIR DOMINIC JOHN CORRIGAN

(1802-1880)

"We know no difference of race, or creed,
or colour, for every man is our neighbour."

—Dominic Corrigan, quoted by Williamson.

DOMINIC JOHN CORRIGAN was born in Dublin on December 1, 1802. His father was a successful farmer who also sold agricultural implements. Young Corrigan was educated at the lay College of Saint Patrick at Maynooth. There he studied the classical languages, French, and natural science. Corrigan distinguished himself in his studies at Maynooth and was frequently called upon to assist the professor of natural philosophy.

Following his schooling at Maynooth, Corrigan was apprenticed to Dr. O'Kelly, physician to the same college. O'Kelly was impressed with Corrigan's ability and advised his father to send the young apprentice to study medicine at Edinburgh University.

O'Kelly's advice was accepted and Corrigan completed his medical studies at Edinburgh. He was graduated with the degree of Doctor of Medicine in 1825. It is interesting to note that he was a classmate of William Stokes, who was also destined to make significant contributions to the study of the diseased heart.

Soon after graduation, Corrigan settled in Dublin where he continued to study as well as to practice medicine. In 1830 he became physician to the Jervis Street Hospital. While at Jervis Street Hospital, Corrigan wrote the two papers that were to make his name famous, "On Permanent Patency of the Mouth of the Aorta, or Inadequacy of the Aortic Valves" (1832), and "On Cirrhosis of the Lung" (1838). In 1831 he was appointed physician to the college at Maynooth. Corrigan was active in the treatment of cholera, that disease having appeared in Dublin in 1832. Elsewhere in the present volume we have related the experience of Stokes, who reported the first case of the epidemic.

Corrigan was an excellent teacher. He became professor of medicine first in the Digges Street School, next in the Peter Street School, and later in the Richmond Hospital (or Carmichael School). An important article by Corrigan, "An Aneurism of the Aorta" was published in the "Lancet" for February 7, 1829. This paper, his first published work, emphasized the value of the stethoscope in the diagnosis of cardiac conditions. His article on aneurysms was soon followed by his "Inquiry into the Causes of 'Bruit de Soufflet' and 'Frémissement Cataire'" published in the "Lancet" for April 4 and April 11, 1829. Therein Corrigan showed that Laënnec's conception of "bruit de soufflet" was erroneous. Laënnec believed that these sounds were the result of spasm, but Corrigan explained them on a purely physical basis. Therein, too, is the first suggestion of what Chauveau and Savart long afterward described as "fluid veins," or eddies in the vessels.

[419]

The immortality of Corrigan, in cardiology, rests of course on his famous paper on aortic insufficiency. This paper it is our privilege to reprint. It was published in 1832 when Corrigan was thirty years of age. When this article was written, Corrigan honestly believed that it was the first published work on the subject. James Hope took violent exception to the assumption and claimed the discovery of aortic insufficiency as his own, made in 1825. Hope claimed that he had taught this subject at St. Bartholomew's Hospital in 1826 and at La Charité, Paris, in 1827.[1] Hope[2] in 1831 had described the ''jerking'' pulse of aortic insufficiency. More noteworthy cases of this condition had been observed earlier than Hope's description, however. William Cowper,[3] in 1705, and Raymond de Vieussens, in 1715, had described the clinical aspects of aortic insufficiency; but they did not attempt to discover the pathologic cause. Samuel Wilks[4] suggested that the most important claim was that of Thomas Hodgkin who, in 1827 and in 1829, had read two papers before the Hunterian Society in which he showed knowledge of the chief signs of aortic insufficiency. However, none of the accounts mentioned equals that of Corrigan for completeness and masterful description.

Another important paper of Corrigan's, published in 1837, in the ''Dublin Journal of the Medical Sciences,'' was ''On Aortitis as one of the Causes of Angina Pectoris.'' It is of interest to note that Rolleston said that Sir Clifford Allbutt, who advanced the same explanation in 1894, did not find out until 1908 that Corrigan had written about it seventy-one years previously.

In addition to ''Corrigan's pulse'' or as the great French clinician, Armand Trousseau, named it, ''maladie de Corrigan,'' another condition of pathologic importance was named after him. This was long known as ''Corrigan's cirrhosis,'' today better known as ''fibroid disease of the lung.'' In his article on cirrhosis of the lung, published in the ''Dublin Journal of Medical Science'' for May, 1838, Corrigan demonstrated the difference between cirrhosis and tubercular phthisis.

That same year (1838) Corrigan became one of the founders of the Dublin Pathological Society. He later served as president of the organization. The publication of these last two papers greatly increased Corrigan's reputation as a clinician, and in 1840 he was elected physician to the Whitworth Medical and the Hardwicke Fever Hospitals. In 1843 he received his diploma as a member of the Royal College of Surgeons (London). On the invitation of the examiners he gave an oral dissertation on the patency of the aortic valves.

In 1849 the honorary degree of Doctor of Medicine was conferred on him by the University of Dublin. With the founding of Queen's University in 1850, Corrigan was made a member of the University Senate. In 1859 he was its representative in the medical council, and in 1871 he was elected vice chancellor of the University.

Corrigan held the office of president of the King's and Queen's College of Physicians for five consecutive years. A colossal statue in white marble of him was made by Foley and it stands next to statues of his great contemporaries, Graves, March and Stokes.

In 1866 Corrigan was created a baronet, not only because of his high attainments in medicine, but also because of his services to the government. In 1870 he was appointed physician-in-ordinary in Ireland to Queen Victoria. That same year he was made one of the commissioners of national education in Ireland.

[1]Dock, George: Dominic John Corrigan, Ann. M. Hist. n.s. 6: 381-395, 1934.
[2]Hope, James: A Treatise on the Diseases of the Heart and Great Vessels, London, 1831, Kidd, 612 pp.
[3]Cowper's contribution is reprinted on pp. 109-114.
[4]Wilks, Samuel: Notes on the History of Valvular Disease of the Heart, Guy's Hosp. Rep. (s.3) 16: 209-216, 1871.

At the age of sixty-eight, Sir Dominic was elected to represent Dublin in the British House of Commons. He held his seat until the dissolution of Parliament in 1874. Corrigan took a great interest in zoology, was a member and later president of the Royal Zoological Society of Ireland, the Royal Irish Academy, the Academy of Medicine of Paris, and of the Harveian Society of London. In 1875 he was elected first president of the Pharmaceutical Society of Ireland.

Corrigan in his later years suffered from attacks of gout. In 1878 he had a slight paralytic stroke and in 1880 he died at the age of seventy-eight following a massive cerebral vascular accident with right hemiplegia.

ON PERMANENT PATENCY OF THE MOUTH OF THE AORTA, OR INADEQUACY OF THE AORTIC VALVES*

By

D. J. CORRIGAN, M.D.

One of the physicians to the Charitable Infirmary, Jervis Street, Dublin; Lecturer on the Theory and Practice of Medicine; Consulting Physician to St. Patrick's College, Maynooth

THE disease to which the above name is given has not, so far as I am aware, been described in any of the works on diseases of the heart. The object of the present paper is to supply that deficiency. The disease is not uncommon. It forms a considerable proportion of cases of deranged action of the heart, and it deserves attention from its peculiar signs, its progress, and its treatment. The pathological essence of the disease consists in inefficiency of the valvular apparatus at the mouth of the aorta, in consequence of which the blood sent into the aorta regurgitates into the ventricle. This regurgitation, and the signs by which it is denoted, are not necessarily connected with one particular change of structure in the valvular apparatus, and hence the name *Permanent Patency of the Mouth of the Aorta, or Inadequacy of the Aortic Valves,* has been chosen as simply expressing such a state of the parts as permits the regurgitation to occur.

I have been in the habit for some years of describing this disease under the name of *Inadequacy of the Aortic Valves;* but as Dr. Elliotson, in his elegantly written work on Diseases of the Heart, has given to a somewhat analogous morbid state of the auriculo-ventricular opening, a better name, *Permanent Patency,* I have, for that reason, and for the sake of uniformity, adopted the term, and I shall continue to use it as synonymous with my own term, *Inadequacy of the Aortic Valves.*

The morbid affections of the valves and aorta permitting this regurgitation are the following.

1st.—The valves may be absorbed in patches, and thus become reticulated and present holes, through which the blood flows back into the ventricle.—*Vid.* Plate I, Fig. 1.

2d.—One or more of the valves may be ruptured; the ruptured valves, when pressed, flapping back into the ventricle instead of catching and supporting the column of blood in the aorta, the blood then regurgitating through the space left by the broken valves.—*Vid.* Plate I, Fig. 2.

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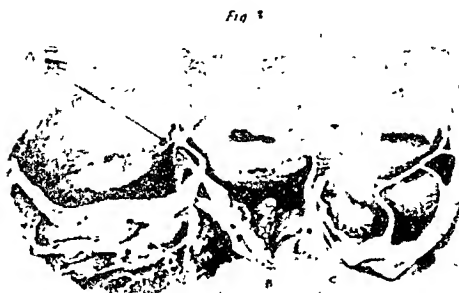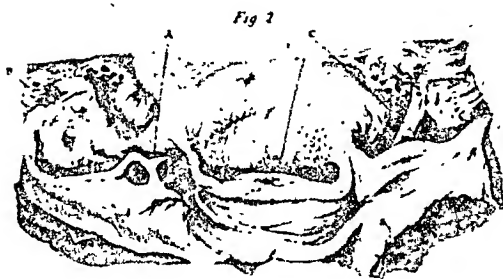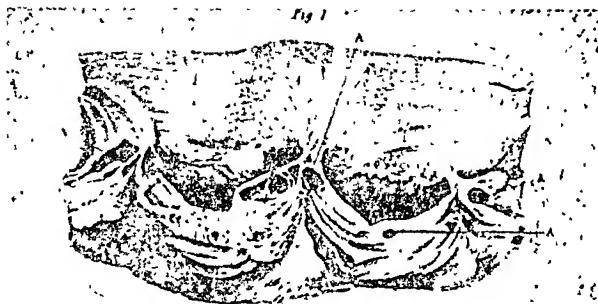
Fig. 1. This figure scarcely needs any explanation.

It shows the reticulated valves.

The letters A, A, A, A, point out the openings produced by absorption in the valves, through which the blood regurgitated. These valves were very slightly thickened.

Fig. 2. A, points out the left hand valve, with an opening through it large enough to admit a goose quill, and ruptured from its connection with the aorta, so that it flapped back into the ventricle.

B. Bony depositions on the inner coat of the aorta.

C. The middle and right hand valves thickened, and contracted in their free edges, so that they could be separated only a very short distance from the sides of the aorta.

Fig. 3. A, A. Openings in the valves, as in Fig. 1, produced by absorption, one of the openings in the right hand valve large enough to permit the finger to pass through.

B. Middle valve, projecting downwards, curled back, and bound to the aorta by bony deposition, so that it was totally useless.

C. Bony deposition tying the edges of the middle and right hand valve together, and at the same time gluing them to the aorta.

3d.—The valves may be tightened or curled in against the sides of the aorta, so that they cannot spread across its mouth; and an opening is then left between the valves, in the centre of the vessel, through which the blood flows freely back into the ventricle.—*Vid.* Plate I, Fig. 3.

4th.—The valves without any proper organic lesion may be rendered inadequate to their function by dilatation of the mouth of the aorta. The aorta, affected by aneurism, or dilated, as it frequently is in elderly persons, about its arch, will sometimes have the dilatation extending to the mouth of the vessel, and in such a case, the valves become inadequate to their function, not from any disease in themselves, but from the mouth of the aorta dilating to such a diameter, as to render the valves unable to meet in its centre; the blood then, as in the other instances, regurgitates freely into the ventricle.

General Symptoms.—On the general symptoms that accompany this disease, little is necessary to be said. Like most of those connected with affections of the respiratory and circulating organs, they are uncertain and unsatisfactory. There are frequently convulsive fits of coughing, more or less dyspnoea, sense of straitness and oppression across the chest, palpitations after exercise, sounds of rushing in the ears, and inability to lie down. Neither one nor all of these symptoms are essential to the disease. They may all arise from varied affections of the lungs, heart, liver, or nervous system. They neither tell us the seat of the disease, nor the extent of the danger.

Signs.—What is deficient in general symptoms from their obscurity, is, however, amply supplied by the certainty of the physical and stethoscopic signs, which may be referred to the three following indications. 1*st*, Visible pulsation of the arteries of the head and superior extremities. 2*d*, *Bruit de soufflet* in the ascending aorta, in the carotids, and subclavians. 3*d*, *Bruit de soufflet* and *frémissement*, or a peculiar rushing thrill felt by the finger, in the carotids and subclavians. In conjunction with these may be reckoned the pulse, which is invariably full. When a patient affected by the disease is stripped, the arterial trunks of the head, neck, and superior extremities immediately catch the eye by their singular pulsation. At each diastole the subclavian, carotid, temporal, brachial, and in some cases even the palmar arteries, are suddenly thrown from their bed, bounding up under the skin. The pulsations of these arteries may be observed in a healthy person through a considerable portion of their tract, and become still more marked after exercise or exertion; but in the disease now under consideration, the degree to which the vessels are thrown out is excessive. Though a moment before unmarked, they are at each pulsation thrown out on the surface in the strongest relief. From its singular and striking appearance, the name of *visible pulsation* is given to this beating of the arteries. It is accompanied with *bruit de soufflet* in the ascending aorta, carotids, and subclavians; and in the carotids and subclavians, where they can be ex-

amined by the finger, there is felt *frémissement,* or the peculiar rushing thrill, accompanying with *bruit de soufflet* each diastole of these vessels. These three signs are so intimately connected with the pathological causes of the disease, and arise so directly from the mechanical inadequacy of the valves, that they afford unerring indications of the nature of the disease. In order to understand their value, it is necessary to consider their connection with the cause by which they are produced. The visible pulsations of the arteries of the neck, etc. may be first examined.

In the perfect state of the mechanism at the mouth of the aorta, the semilunar valves, immediately after each contraction of the ventricle, are thrown back across the mouth of the aorta by the pressure of the blood beyond them, and when adequate to their function of closing the mouth of this vessel, they retain in the aorta the blood sent in from the ventricle, thus keeping the aorta and larger vessels distended. These vessels consequently preserve nearly the same bulk during their systole and diastole. But when the semilunar valves, from any of the causes enumerated, become incapable of closing the mouth of the aorta, then after each contraction of the ventricle, a portion of the blood just sent into the aorta, greater or less, according to the degree of the inadequacy of the valves, returns back into the ventricle. Hence the ascending aorta and arteries arising from it, pouring back a portion of their contained blood, become, after each contraction of the ventricle, flaccid* or lessened in their diameter. While they are in this state, the ventricle again contracts and impels quickly into these vessels a quantity of blood, which suddenly and greatly dilates them. The *diastole* of these vessels is thus marked by so sudden and so great an increase of size as to present the visible pulsation which constitutes one of the signs of the disease.

That this visible pulsation of the arteries is owing to the mechanical cause here assigned is made evident by several circumstances. It is most distinct in the arteries of the head and neck, which empty themselves most easily into the aorta, and of course, into the ventricle. In the arteries of the lower extremities of even larger size than those which present it about the head and neck, it is not seen to any comparative degree, and most generally not at all while the patient is standing or sitting. It is much more marked in the arteries of the head and neck in the erect than in the horizontal posture; and a patient suffering under the disease himself, first pointed out a circumstance which is convincing of its being produced as asserted. He could increase the pulsation of the brachial and palmar arteries in a most striking degree by merely elevating his arms to a perpendicular position above his head. He thus enabled the brachial and palmar arteries to empty themselves more easily back upon the aorta. They became more flaccid, and

*It may be objected to the phrase *flaccid,* that the arteries, being capable of contracting upon whatever quantity of blood they may contain, are never flaccid. In using the phrase, it is not meant that the sides of the arteries, like a collapsed vein, fall together, but merely that, having become emptied of some of their blood, in consequence of its regurgitation into the ventricle, they are, while in this state, less tense than when at the next diastole they are distended by a fresh supply of blood to their limit of extension.

then, on the next contraction of the ventricle, their diastole became comparatively greater, and their visible pulsation of course more marked. The same effect could be produced in the arteries of the lower extremities by lying down and elevating the legs on an inclined plane. The strength of the heart has little to do in producing this singular pulsation, for it is never observed in an equal degree, and most generally not at all, in the arteries of the lower extremities.

If it be asked, is the explanation here adduced of the cause of this visible pulsation sufficient to account for its appearance in the brachial and radial arteries, since the blood to return back from these vessels into the arch of the aorta should flow upward when the patient holds his arms in the ordinary position, flexed or by hanging by his side? The following reply may be made. When the subclavians are pouring back their blood into the arch of the aorta and ventricle, the elasticity of the brachial arteries, acting upon the blood just urged into them, forces it back along with the retrograde current of the subclavians, no obstacle meeting it in that direction. The brachial arteries thus partially empty themselves, and become in their systole of a lessened diameter like the carotids and subclavians, but in less degree. The next jet of blood from the ventricle dilates them, and as in the subclavians, produces in them a visible pulsation; and if they be assisted in returning their blood by elevating the arms to a perpendicular position, their pulsation becomes, as has been already observed, much more strongly marked. The arteries of the lower extremities are not similarly circumstanced. The arteries of the upper extremities are assisted in emptying themselves back towards the heart, by the retrograde current in the subclavians and ascending aorta; but on the blood contained in the arteries of the lower extremities, the tall column of blood in the descending aorta is pressing, and prevents any return; or if it be supposed that of the large mass of blood in the descending aorta, a small portion flows back into the arch, it can produce little change in the contents of the iliacs and femorals; and moreover, whether the column of blood in the aorta be lessened or not in diameter, the pressure on the contained blood of the iliacs and femorals will remain the same, and keep these vessels distended. If we, however, as already observed, after the relation of the several arteries to the arch of the aorta, so as to facilitate the reflux of their contained blood, for instance from the radial arteries, by raising the arms to a perpendicular line above the head, from the iliacs and femorals, by placing the patient in a recumbent posture, and raising the legs upwards on an inclined plane, the visible pulsation becomes much more marked in these respective arteries.

The *bruit de soufflet,* which is heard in the ascending aorta, carotids, and subclavians, with the accompanying *frémissement* in the latter arteries, is next to be considered. The *bruit de soufflet* characterizing this disease, is heard, as already observed, in the ascending aorta, its arch, and in the carotids and subclavians. It can be followed upwards from the fourth

rib along the course of the aorta, increasing in loudness as it ascends, until it is heard of great intensity at the upper part of the sternum, where the arch of the aorta most nearly approaches this bone, and then branching to the right and left, it can be traced into the carotids and subclavians of both sides; and in these trunks it assumes a harshness that it did not possess in the aorta. This *bruit de soufflet* is synchronous with the visible pulsation, with the diastole of the arteries. It is no consequence whether the ascending aorta and its large branches be sound or be diseased; the *bruit de soufflet* is as loud in the one case as in the other. To account for the presence of this sign, and why it extends so far from the seat of the disease and along sound vessels, it is necessary to refer to a paper published in the *Lancet* of 1829, Vol. II, p. 1.[*] Continued observations from the date of that paper to the present, have confirmed the view then taken of the cause of that singular sound; of its being dependent, purely on a physical cause, on a mechanical change in the manner of the blood's flowing.

In that paper is related an experiment, which it may be well to recapitulate here. A flexible tube, such as a piece of small intestine, or a portion of artery, is connected by one end with a tube which has a current of water of considerable force running through it. While the piece of intestine or artery is kept fully distended by the supply of water from the tube, no sound is produced by the motion of the fluid; but if the flexible tube, while the fluid is moving through it, be pressed upon in any part, so that the quantity of fluid passing through the contracted part is no longer sufficient to keep the further portion of the tube tense, then, beyond the contracted part, where the tube is less tense, or in some degree flaccid, a distinct, and, according to the velocity or force of the current, a loud *bruit de soufflet* is heard; and, at the same time, if the finger be gently laid upon the part of the tube where the *bruit de soufflet* is heard, a slight trembling of the tube is perceived, evidently arising from the vibrations into which the current within is throwing its sides. If, in place of constricting any one part of the flexible tube, the whole tract of tube be allowed to become partially flaccid, by diminishing the supply of fluid, and the fluid be then allowed to rush along the tube by jets, at each jet the tube is suddenly distended, resembling the visible pulsation described above; and with each diastole of the tube, there is a sudden and loud *bruit de soufflet;* and, synchronous with the *bruit de soufflet,* there is *frémissement* felt by the finger.

Both the sound heard and the sensation felt by the finger in this experiment may be explained by the principles which regulate the motion of fluids. It may be remarked, that it is a property of fluid in motion, that, when discharging itself from the orifice of a tube into open space, or into a vessel of wider capacity not fully distended, its particles move in lines from the orifice, like so many *radii* tending to leave vacuums between them. When the flexible tube, artery or intestine, therefore, is kept fully dis-

*Corrigan, D. J.: Aneurism of the aorta; singular pulsation of the arteries, necessity of the employment of the stethoscope, Lancet 1: 586–590, 1829.—F.A.W., 1940.

tended, the fluid moves forward as a mass, there is no tendency in its particles to separate from one another,—they all press equally,—there is no vibratory motion of the sides of the tube, and consequently no sound, and no *frémissement* or trembling. But if the tube be not kept fully distended, then the fluid propelled through it rushes along as a current; and its particles tending to leave vacuums between them, throw the sides of the tube into vibrations, which can be very distinctly felt by the finger, and which give to the ear the peculiar sound *bruit de soufflet,* and to the touch *frémissement.*

These principles may be applied to the state of the ascending aorta and its branches in the instances before us. When the aortic valves are fully adequate to their function of perfectly closing the mouth of the aorta, and thus preventing any regurgitation of blood, the aorta and its branches are kept fully distended, the blood is at each contraction of the ventricle propelled forward *en masse,* and there is no trembling, or vibratory motion of the sides of the aorta, carotids, and subclavians, and, as in the flexible tube when fully distended, no sound is emitted. But when the valves, becoming inadequate to their office, permit some of the blood contained in the ascending aorta, carotids, and subclavians, to return into the left ventricle after each contraction, then the aorta and these trunks become, like the flexible tube in the second part of the experiment partially flaccid; and at the next contraction of the ventricle, the blood propelled into them is sent along as a rushing current, which throws the sides of these arteries into vibrations, and these vibrations give to the ear *bruit de soufflet,* and to the finger *frémissement.* These two signs may be traced to a varying distance from the mouth of the aorta, and always along the carotids, and to the outer third of the subclavians, and sometimes in the brachial arteries, as far as the bend of the arms, the distance to which they are heard being determined by the limit to which the current-like motion of the blood producing them is extended. In those cases in which the deficiency of the valves is considerable, allowing a full stream of blood to rush back into the ventricle, there is heard in the ascending aorta a double *bruit;* the first accompanying the *diastole* of the artery, the second immediately succeeding; and, in listening to the two sounds constituting this double *bruit de soufflet,* the impression made distinctly on the ear is that the first sound is from a rushing of blood up the aorta, the second from a rushing of it back into the ventricle. It is impossible for those who have not heard this double *bruit* to conceive the distinctness with which the impression described is made on the ear. A patient in one instance heard this double sound distinctly in his own person, and referred it to its cause, a rushing of blood *from* and *to* the heart. The *bruit de soufflet* and *frémissement* are not perceived in the arteries of the lower extremities, when the patient is in a sitting or standing posture. The pressure of the blood in the abdominal aorta is sufficient in these postures to keep the vessels arising from it fully dis-

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tended; and thus no vibratory motion of their parietes being permitted, there is no bellows sound; nor *frémissement* or rushing thrill.

History and Progress of the Disease.—Of eleven cases of the disease, only two occurred in females, and in both of these the valves were nearly quite sound in texture; but the aorta being thinned and dilated, the valves could not meet so as to prevent regurgitation. None of the cases occurred in very early age. The youngest person presented labouring under the disease was twenty years of age. In this respect, inadequacy of the aortic valve differs from narrowing of the left auriculo-ventricular opening, which is not unfrequently met with in children, and even in infants at the breast. The causes of the disease are uncertain. In one case the disease followed an attack of acute rheumatism, which had been accompanied with symptoms of *pericarditis.* In some cases the commencement of the disease was referred by the patient to an inflammatory affection of the chest, which had occurred months or years before; while in others no cause or date could be assigned.

The symptoms accompanying its commencement and progress are very variable. Most generally the patient describes the first sensations as having been a feeling of oppression and straitness across the chest, with palpitation of the heart on any unusual exercise. These symptoms become gradually more distressing, and are after a very uncertain period of time accompanied by fits of coughing resembling paroxysms of asthma, and terminating in scanty expectoration. In a few cases, however, cough was not at any time, even up to the last hours of life, an urgent symptom; the oppression and straitness of the chest, with palpitation on any exertion, and an anxiety for a supply of fresh air, being the principal complaints. As the disease proceeds, the straitness and oppression about the chest become more distressing; fits of coughing more frequent; and the patient has an anxiety, approaching to agony, for a free supply of fresh air, frequently starting from bed at night under the dread of suffocation. In the last stage the state of suffering is extreme. The patient will not lie down for a moment from the dread of suffocation. The face, which had been pale, becomes purple on the lips as in suffocative catarrh; oedema of the legs comes on, followed ultimately by oedema of the hands and arms; there is no sleep, or there are almost incessant startings from it; the countenance assumes a most painful expression of sinking; and the patient at length dies exhausted. The pulse in no case was under eighty. It ranged from that to 110; and in every case it has been all through the disease (unless influenced by medicine) full and vibrating, even to within a few hours of death. In the course of the disease, the superficial branches of the carotids, the brachial arteries, the radial, and the ulnar, and their branches, wherever near enough to the surface to be traced, become apparently enlarged, and remarkably tortuous;—the brachial artery in parts of its course often almost doubling upon itself. The *frémissement,* or rushing thrill, described as easily felt in the subclavians

and carotids, can sometimes be felt by moderate tact as fast as the pulse in the wrist. The heart in all the cases that occurred was enormously enlarged, and its bulk arose from the state of the left ventricle, which in some cases was so much enlarged in cavity and in thickness, as to make the organ resemble rather the heart of a bullock than that of a man. The other parts of the heart, although necessarily obliged to keep pace in some measure with this increased size, did not at all partake equally in the enlarged bulk. The impulse of the heart was far less than natural, even in cases where the hypertrophy of the left ventricle was greatest. In some of them no impulse could be felt; and in none did the impulse during life give at all a proportional measure of the excessive hypertrophy discovered after death.*

Haemoptysis very rarely occurs in the course of the disease, and the lungs are generally found after death permeable to air, and remarkably healthy. This is owing to the sound state of the auriculo-ventricular opening of its valves. This opening being full sized, permits the blood to pass with freedom into the ventricle, where it is retained by the sound auriculo-ventricular valves; and thus those sudden congestions of the blood vessels of the lungs, so common in narrowing of the left auriculo-ventricular opening, are remarkably rare. The manner of death in inadequacy of the aortic valves is different from that in narrowing of the auriculo-ventricular opening. In the latter, owing to the obstacle presented by the narrowed opening to the passage of blood into the ventricle, the lungs are by any slight exciting cause suddenly congested; and the patient dies, not from the direct effect of the organic affection of the heart, but from the superinduced affection of the lungs,—pulmonary apoplexy, pneumonia, or suffocative catarrh. In the disease under consideration, the patient appears to die of mere exhaustion. The inefficiency of the valves of the aorta throws a great increase of labour on the left ventricle. The muscular energy of this part of the heart is in the course of time worn out. The heart is at length incapable of sustaining the column of blood incessantly pressing upon it; it ceases to contract and is found after death largely distended with blood. The symptoms preceding death are in accordance with this state. For some days, or even

*Laennec has stated, and his assertion is supported by many, that the degree of impulse is always a correct index of hypertrophy of the ventricle, but it is now admitted by some most capable of judging, that the impulse of the heart is not to be considered a gauge of the hypertrophy of the ventricle. Andral, in his *Clinique Medicale*, Vol. II, p. 160, says, "Plus d'une fois dans des cas où après la mort nous avons trouvé les parois des ventricules très epaissies en même temps que leurs cavités étaient notablement agrandies, nous n'avions reconnu pendant la vie aucune espèce d'impulsion. Dans d'autres cas, ou il y avait simple hypertrophie due ventricule gauche, avec grande diminution de sa cavité (hypertrophie concentrique de MM. Bertin et Bouillaud,) il n'y avait pas eu non plus d'impulsion appréciable." Piorry (*Sur la Percussion*, p. 139) says, that impulse of the heart, carried even to raise the head of the observer, is far from being a constant sign of hypertrophy. Dr. Graves, in a clinical lecture (*vid. Med. Gazette, March, 1831, p. 714*,) says, "I can assert in the most positive manner, that I have seen cases of pneumonia in which the heart's pulsation continued violent until within a short time of dissolution; so much so indeed, as to induce the erroneous belief in myself and other medical attendants, that this organ was in a state of hypertrophy and dilatation, and yet it was found after death to be in every respect healthy." This subject will be resumed at another time.

weeks, before death, nature appears to be struggling against overwhelming exhaustion. The patient is constantly in the most heart-rending tone imploring to be relieved of the weight that is upon him; the countenance expresses the greatest sinking and distress; there are anxious calls for fresh air and a continual restlessness, similar to what is seen in a patient sinking from hemorrhage; and when in this state the patient in some trifling motion dies exhausted.

The duration of this disease is very uncertain. No case was of less duration than two or three years, and some of the cases at present under treatment have been of seven or eight years standing. The time during which the disease may continue without terminating fatally, seems to depend principally upon the extent to which regurgitation is permitted. The cases in which the valves, from small perforations, allowed but little regurgitation continued for many years; while the case which furnished the Plate No. II and in which the valves were ruptured and much injured, allowing considerable regurgitation, terminated fatally in less than three years.

Diagnosis.—Inadequacy of the aortic valves may be confounded with narrowing of the mouth of the aorta, either congenital or from diseased valves, with disease of the auriculo-ventricular valves, with aneurism of the arch of the aorta or *arteria innominata*, with nervous palpitations, and with asthma. Congenital narrowing of the mouth of the aorta is a very rare disease, but narrowing of the mouth of this vessel produced by vegetations on the valves is not unusual; and *bruit de soufflet* is a sign common to it, and to the disease we are considering. The resemblance between the signs of the two diseases extends, however, no farther. The visible pulsation of the arteries, arising from the arch of the aorta, which forms so striking a sign of inadequacy of the aortic valves, is wanting in narrowing of the mouth of the aorta. The pulse also is strikingly different in the two diseases. In narrowing of the aortic orifice it is small and contracted; in inadequacy of the aortic valves it is invariably full and swelling. In narrowing of the aortic orifice there is generally a marked contrast between the pulse and the impulse of the heart. The pulse is small and contracted; the impulse of the heart is strong and energetic. In the disease we are considering, when there is a contrast it is always in the inverse way; for while the arteries beat with violence, and the pulse is strong and full, the impulse of the heart is scarcely perceptible. When the mitral valves, becoming indurated or ossified, produce narrowing of the auriculo-ventricular opening, that narrowing produces *bruit de soufflet;** and the *bruit de*

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*The principles regulating the motion of fluids, already laid down, explain the production of *bruit de soufflet* in narrowed auriculo-ventricular opening. The blood at each contraction of the auricle discharges itself from a narrow orifice into the ventricle, "a vessel of wider capacity not fully distended."—"The particles" of the blood "move in lines from the orifice, like so many radii tending to leave vacuum between them." This motion, as in the experiment of the tube, throws the sides of the ventricle into vibrations, which produce on the ear *bruit de soufflet*, and if the heart thus affected come forward so as to transmit through the parietes of the chest this vibrating motion, the hand laid over the heart perceives a *frémissement*, or trembling in the organ, the *bruissement* of Corvisart.

soufflet thus produced might be confounded with that accompanying inadequacy of the aortic valves. Independently of the visible pulsation of the arteries, and the state of the pulse, which accompany inadequacy of the aortic valves, stethoscopic examination points out with certainty the distinction of the two diseases. When the *bruit de soufflet* is produced by narrowing of the auriculo-ventricular opening it is heard loudest just where the impulse of the heart against the side is felt; it comes with the impulse, and if loud it seems to rush into the ear; and as the stethoscope is removed from this point upwards along the sternum, it is heard growing fainter the farther the point of examination is from the point where the impulse is felt. In inadequacy of the aortic valves, the converse holds. For over the point where the impulse is or should be felt, *bruit de soufflet* is either not heard at all, or heard very indistinctly; but as the stethoscope is moved upwards from the heart, in a line corresponding with the ascending aorta, the *bruit de soufflet* is heard growing louder and louder, until over the arch of the aorta, and in the large trunks arising from it, the sound grates upon the ear with harshness.

Permanent patency of the mouth of the aorta may be mistaken for aneurism. If the arch of the aorta and *arteria innominata* approach more nearly than usual to the notch of the sternum, the visible pulsation at the root of the neck becomes so prominent, as to lead to a supposition that there is aneurism, and even of considerable size at this part.

Very lately a case came under my observation, in which there was a remarkable resemblance to aneurism. So strong were the pulsations for years in the region of the *arteria innominata*, that until the examination after death there was never even a doubt expressed that the case was not aneurism. The aorta was thinned, and was dilated so much as to render the valves inadequate to their office, and leave a permanent patency between them. The *arteria innominata*, the carotids, and subclavians, were also dilated beyond their natural size, thus increasing the appearance of the pulsation, but there was no trace whatever of aneurism in the *arteria innominata*, such as had been supposed to exist there during life. An acquaintance with the disease under consideration, and a knowledge of the fact, that a violent throbbing at the root of the neck, or notch of the sternum, may arise from another cause than aneurism, will prevent the forming of a rash opinion on the cause of the violent throbbing. This throbbing may proceed from aneurism, or may arise from inadequacy of the aortic valves. When it proceeds from aneurism of the arch, or of the *arteria innominata*, it is confined to the vessel or the region of the vessel affected; the other trunks arising from the arch present only their natural, or even a diminished pulsation, and there is in the trunks arising from the arch neither *bruit de soufflet* nor *frémissement*. On the contrary, when the throbbing at the notch of the *sternum*, or in the region of the *arteria innominata* is from inadequate aortic valves, all the larger trunks arising

from the arch pulsate in an equal degree, or with trifling differences, arising merely from the relative sizes of the vessels, or their relation to the surface, and they are never at any time without *bruit de soufflet* and *frémissement*.

Not only in relation to treatment, but in regard to the patient's mental anxiety, it is of importance to be aware, that inadequacy of the aortic valves may, by this violent pulsation at the root of the neck, closely simulate aneurism of the arch of the aorta, or the root of the *arteria innominata*. In aneurism of the aorta life is not for a moment secure, and it may be necessary that even for a remote hope of cure the patient should totally abstain from all exertion. In permanent patency of the mouth of the aorta the fatal result is never sudden; and under proper restriction the patient is not only able to lead an active life for years, but is actually benefited by doing so.

The two diseases, aneurism of the aorta, and inadequacy of the valves, may, however, be combined. Aneurism of the ascending aorta may, by extending to the mouth of this vessel, dilate it so, that the valves are unable to meet, and there is then a combination of the two diseases; there is aneurism and there is permanent patency of the aortic opening. The first cases that came under my observation presenting the signs of inadequacy of the aortic valves were cases in which the valves were rendered useless in this way, namely, by the mouth of the aorta sharing in the aneurismal dilatation. These cases led me into an error; for, meeting the signs of permanent patency of the aortic orifice in conjunction with aneurism, I erroneously attributed to the aneurism the signs which arose from the permanent patency.* Aneurism of the aorta of itself does not produce the signs arising from permanent patency of the mouth of the aorta. It can only produce them in the way already described, by involving in the dilatation the mouth of the aorta; and hence, when in conjunction with an aneurismal tumour of the *arteria innominata* or aorta, there are found visible pulsation, *bruit de soufflet*, and *frémissement* in the ascending aorta, and the trunks arising from it, we may be certain, that, in addition to the aneurism, there is a defect in the aortic valves, or that the aneurism has extended downwards, involving the mouth of the aorta. On the other hand, if these signs be absent, the valves are sound, and the mouth of the aorta is not included in the disease. The propriety of performing Mr. Wardrop's, or indeed the common operation for aneurism about the neck, might depend on the information thus acquired of the state of the aortic valves. To perform either in a case where the aneurismal dilatation was so extensive as to involve the mouth of the aorta, or where the aortic valves were diseased, would only bring the surgical treatment of the disease into un-merited discredit.

Palpitation of the larger arterial trunks, depending on derangements of the nervous system, will sometimes in their violence simulate the visible

Vide Lancet for February 7th, 1829.

pulsation arising from inadequate aortic valves; and in females these palpitations will last not only for months but for years, and seem to justify an opinion that there is organic disease of the heart. This nervous palpitation is not, however, accompanied by *bruit de soufflet* and *frémissement;* and the absence of these two signs is conclusive as to the nature of the disease. Sometimes, however, more than one examination is required before pronouncing a positive opinion; for in a nervous patient, the alarm excited by the first examination will render the circulation hurried and irregular, and hence there may be in the carotid or subclavian a momentary *bruit de soufflet.* In making the examination it is moreover necessary, that the edge of the stethoscope should not be allowed to press on the artery, because its pressure is sometimes sufficient in those cases to produce the sound. When the *bruit de soufflet* and *frémissement* are only momentary, no value should be attached to them. In permanent patency of the aorta they are never absent. The convulsive fits of coughing ending in difficult mucous expectoration have made some cases of this disease be mistaken for asthma, and the state of the pulse has served to maintain the error; for the pulse being remarkably full, as it always is in the disease we are considering, seemed to be sufficient evidence that there was in the heart no obstruction to the circulation; hence the convulsive fits of coughing were supposed to have their origin in the lungs. With a knowledge of the signs afforded by the disease, no one of even moderate acquaintance with the stethoscope can confound it with asthma; without a knowledge of the stethoscope it will, however, be impossible in very many instances to distinguish between the two diseases. General symptoms will give no information on which the slightest reliance can be placed.

Treatment.—There is no class of diseases to which the scientific principles that guide modern medicine have been less applied than to diseases of the heart. From its curious mechanism, from the varied derangements to which that mechanism is subject, from the number of tissues that enter into its formation, and from its numerous sympathies, its diseases frequently demand most opposite lines of treatment; and yet, it would seem, from the perusal of works on the subject, that one principle were thought sufficient for guiding the treatment of nearly all the diseases of this important organ. With the idea of heart disease, is too frequently associated the notion that such disease, without regard to its precise nature or its cause, requires the action and continued enforcement of measures calculated to exhaust strength and depress vital energy; and this error is sanctioned by the standard works on the treatment of heart disease.

Corvisart says, that "in a great number of organic lesions of the heart, as, for example, in active aneurism, the indication is to diminish the general strength of the patient, and that of the heart in particular." Laënnec, p. 739, says that "though we cannot remove indurations of the valves and narrowing of their openings, we are nevertheless in such

cases to follow up the same measures (bleeding and starving), to remove or diminish hypertrophy;" and Bertin, p. 233, states that "the treatment of valvular alterations is to consist of general and local bleedings, of low diet, of preparations of digitalis," etc.; and, p. 367, "that the measures to be employed against hypertrophy are to be essentially antiphlogistic, and calculated to produce debility." A little reflection on the nature of the disease now before us will show that these principles are inapplicable both to the treatment of the valvular alterations, and of the hypertrophy of the left ventricle, which accompanies that alteration.

The disease we are considering is an inadequacy in the valvular apparatus at the mouth of the aorta permitting a regurgitation of blood into the ventricle. In the perfect state of the valvular apparatus at the mouth of the aorta, the valves support by intervals the column of blood in the aorta, and the heart with its ordinary complement of fibre and of muscular strength, is with this assistance competent to the office it has to perform. But when, in consequence of a deficiency in the valvular apparatus, the heart does not receive its due share of assistance from these valves, and is obliged to perform not only its own function of propelling the blood, but has in addition to support after each contraction a portion of that weight of blood which should then be wholly supported by the valves, it is no longer in its ordinary state equal to the task imposed upon it. In such circumstances, nature, to enable the heart to perform the additional labour thrown on it, increases its strength by an addition of muscular fibre, and the heart thus becomes hypertrophied, in accordance with the general law, that muscular fibres become thickened and strengthened when there is additional power required from it. Is this hypertrophy disease, or is it a wise provision of nature, by which the organ is thus made equal to the increased labour it has to perform? On the answer depends the treatment to be adopted; and on this there is no room for hesitation. A heart of ordinary strength could not, under the circumstances, carry on the circulation; and nature then wisely endows the heart with the requisite degree of strength. It is at once obvious that to interfere with this wise provision of nature, to diminish the strength of the heart, or, if we choose other words, to direct, according to the advice of Laënnec, Bertin, etc. our measures against the hypertrophy of the organ, is to deprive the system of the only power which enables the heart to carry on the circulation. No one thinks of directing measures to diminish hypertrophy of the muscular tissue of the stomach, in narrowing of the pylorus from scirrhus of the bladder or rectum in stricture of the urethra or intestines.

In these instances the hypertrophy is recognized as a provision of nature to make the power of the part equal to the obstacle it has to overcome; and yet, this simple principle seems to have been entirely overlooked in diseases of the heart, as if this organ possessed muscular fibres

of a different nature from other organs, or as if, in adapting itself to obstacles affecting its action, it follows laws different from other muscular parts. The consequence of the neglect of this principle has been, that too often, in treatment of a valvular alteration in the heart, there has been a constant struggle between nature and medicine. Nature has been making the organ equal to its task; while medicine has been directed to counteract nature's efforts, and, by weakening the organ, to render it totally incapable of its task. The repeated bleedings, the starvings, the enforcement of debilitating measures, are totally unsuited to the disease we are considering.

Instead of such treatment, the measures most beneficial are those which by strengthening the general constitution, will give a proportionate degree of vigour to the muscular power of the heart, and thus enable it to carry on the circulation in the absence of that assistance which it ought to receive. With this view, a generous and sufficient diet of animal and vegetable food should be advised, at the same time that an abstinence from those beverages, such as malt liquors, which increase much the mass of the fluids, should be enjoined. It is not at all necessary that the patient should be prohibited from attending to his business or profession, provided that he do not devote to it so much attention as to produce debility. And as there is among patients who have learned that they are afflicted with heart disease an universal dread of sudden death, it is necessary to undeceive them on this point; and in the present instance it can be done with perfect safety, as the termination of the disease is never sudden. This plan of treatment, opposite to what has been generally enjoined, was forced upon the attention long before the reasoning adduced here had been brought to support it.

One case may be mentioned, out of many that occurred, showing the bad effects of debilitating treatment in the disease before us, and exemplifying the evil of acting as if one principle were sufficient for guiding us in the treatment of all heart diseases. It is now several years since a consultation was held upon the case alluded to. This treatment ordered was in accordance with that generally recommended, consisting of repeated small bleedings, blistering, the exhibition of digitalis, and the most rigid regulation of diet, a total abstinence from animal food, and even a spare allowance of vegetables and milk. At the time the patient, a young man, was put under this treatment, he was not in an alarming state; but the disease being recognized as heart disease, he had the fortitude to submit to a course which he was led to expect held out a prospect of cure. Bleeding after bleeding, and blister after blister, were repeated, starvation enforced, and digitalis exhibited, until the patient was reduced to such weakness that he had scarcely strength to raise himself in bed. The local disease was all this time, however, growing worse; for the

palpitation, cough, etc., were, from the slightest cause, increased to greater violence than previously to the commencement of treatment. The plan was, nevertheless, persevered in, until the patient's death being supposed at hand, this debilitating treatment was discontinued. From that hour the patient got better; and as muscular strength returned, the embarrassment of the breathing, palpitation, cough, etc., became less and less urgent. The patient is still alive, the disease is still present; but, with full living and good air, he is able not only to take considerable exercise, but even to undergo the fatigue of a business that constantly requires very laborious exertion.

Having laid down the plan of treatment proper to be adopted as far as it produces effects upon the system, and through it upon the heart constituting a part of the system, it now remains to examine the propriety of employing in this disease a remedy such as *digitalis*, which produces a specific effect upon the heart, rendering its action slow and weak, and which in consequence of that effect is usually recommended in cases of heart disease in conjunction with the measures already deprecated. In inadequacy of the aortic valves the pulse generally ranges from 90 to 110. After each contraction of the ventricle during the pause or interval of rest occurring between that contraction and the next following, a quantity of blood is regurgitating into the ventricle. The danger of the disease is in proportion to the quantity of blood that regurgitates, and the quantity that regurgitates will be large in proportion to the degree of inadequacy of the valves, and to the length of pause between the contractions of the ventricle during which the blood can be pouring back. If the action of the heart be rendered very slow, the pause after each contraction will be long, and consequently the regurgitation of blood must be considerable. Frequent action of the heart, on the contrary, makes the pause after each contraction short; and in proportion as the pauses are shortened, the regurgitation must be lessened. Instead, then, of regarding an increase of frequency in the action of the heart as an aggravation of the disease, it must be viewed, as we have already viewed hypertrophy of the heart, as a provision for remedying as far as possible the evil consequences arising from inadequate valves. To retard in such circumstances the action of the heart would be to do an injury. In every case of this disease in which *digitalis* has been administered, it has invariably aggravated the patient's sufferings. The oppression has become greater; the action of the heart more laboured; the pulse intermittent, and very often dicrotic, from the heart's being unable by a single contraction to empty itself; general congestion and dropsy, if present, have been increased, and in some of the instances *bronchitis* from congestion has been induced; the respiration became laborious, and the strength so much sunk, that patients seemed almost moribund. From this state they only recovered by omitting the *digitalis,*

cidentally occur in cases of inadequacy of the aortic valves, should be opposed with promptitude and decision; bleeding, when used, should be large; but when the inflammatory affection is once subdued, we should cease as soon as possible from debilitating treatment,—which, if persevered in, will prove injurious to the organic affection. No details need be given of the measures to be adopted in those incidental affections, because they differ in no respect, except in energy from the usual treatment for the affection, whatever it may be, that has chanced to supervene. This active treatment on the occurrence of local inflammation is not at all incompatible with the course recommended to be followed when there is no disease present but the valvular inadequacy. Those incidental inflammatory or congestive affections will be rendered less likely to occur by the previous course of management recommended; for the greater vigour the system has enjoyed, the less danger there is of slight causes producing inflammation or congestion.

Lastly, there is besides the supervention of local inflammation or congestion, yet another circumstance, in which blood-letting may be required. Without the occurrence of any apparently adequate cause, straitness of chest, difficulty of breathing, tumultuous action of the heart, and a general feeling of nervous oppression are complained of. Neither pneumonia nor inflammatory action in any organ can be detected, and these symptoms seem to arise from an increase of bulk in the absolute mass of blood circulating, which keeps all the vessels so distended that the heart becomes oppressed, incapable of freely contracting, and tumultuous in its action. Relief is at once afforded by a large blood-letting, speedily followed by the exhibition of a full dose of an opiate. For the employment of the opiate, I am indebted to the suggestion of my colleague, Dr. Hunt, and its good effects are such as to have no substitute for it. The employment of a large bleeding in either of the circumstances here detailed, is very different from the repetition of those irritating small bleedings that are usually practised.

Fits of coughing are sometimes very troublesome in the course of the disease, and where they arise from trifling bronchitis, they are best relieved by pectoral mixtures with a large proportion of opium, not less than four or five grains to an eight ounce mixture.

In these observations no medicine or treatment has been recommended with the view of acting directly on the aorta or valves, so as to restore in any degree the function of the latter. There is no medicine that can have any such power after the disease has been of much standing; and if the valves have become perforated or broken, it is obviously impossible to restore them to their original state. The disease is seldom seen in the commencement. Perhaps if seen early in those cases where it has followed an attack of rheumatism or where it partakes of an inflammatory

character, the employment of mercury pushed to salivation, and counter-irritation, might check the progress of the disease. In the advanced stages no good effect on the valvular affection has been produced by any or all of these measures.

Although the cure of *Inadequacy* of the Aortic Valves is probably out of the reach of medicine, a correct knowledge of the nature of the affection is not the less necessary. The patient is relieved from harrassing treatment, that, however, applicable in other cases of heart disease, is not alone useless, but positively injurious in this. In other affections of the heart there is a constant danger of sudden death from pulmonary apoplexy or hemorrhage, which may be induced even by ordinary exertion, and such danger keeps the patients in a state of perpetual terror. In this disease, on the contrary, assurance may be given against any sudden termination; and the patient may be permitted not only to attend to his business or profession, but may be assured, that, in leading a life of business and tolerable activity, he is adopting the very best means to prolong his life. Under treatment such as recommended, it is astonishing what little uneasiness inadequacy of the aortic valves will produce,—indeed, very often not so much as those organic affections or growth of the liver, which are nevertheless viewed by the profession and by patients with much less terror.

13, Bachelor's Walk, Dublin.
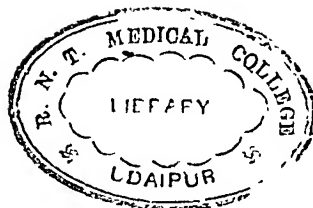
1835

JEAN BAPTISTE BOUILLAUD

THE PATHOLOGY OF ENDOCARDITIS

JEAN BAPTISTE BOUILLAUD
Portrait by C. H. Lehman, 1875

(Courtesy Charles C Thomas.)
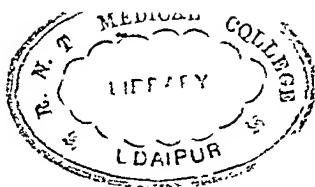
JEAN BAPTISTE BOUILLAUD

(1796-1881)

ON SEPTEMBER 16, 1796, Jean Baptiste Bouillaud was born at Bragette, near Angoulême in France. He attended the Lycée d'Angoulême and distinguished himself by winning the "prix d'excellence." He also received a prize at this school for a poem written in Latin.

Encouraged by his uncle, a surgeon-major in the French army, Bouillaud decided to become a member of the medical profession. He therefore left his native town to study medicine at Paris. His studies were interrupted by the march of the Allies on Paris. Part of Napoleon's army under Marshal Auguste Marmont (1774-1852) and Marshal Édouard Mortier (1768-1835) unsuccessfully attempted to withstand the Allies. Bouillaud joined the students of the École Polytechnique in this ineffectual resistance. Later, when Napoleon returned from Elba, Bouillaud, who had great admiration for the Corsican, enlisted in a Hussar regiment. After the defeat of Waterloo, Bouillaud resumed his medical studies.

It was Bouillaud's good fortune to have many illustrious teachers. Among the most prominent were Guillaume Dupuytren (1777-1835), whom Bouillaud attended in the master's final illness. Dupuytren left special instructions that Bouillaud should perform the necropsy. His other teachers included François Joseph Broussais (1772-1838), of whose blood-letting theories, unfortunately, Bouillaud was a fervent disciple, Jean Nicolas Corvisart (1755-1821), under whom, no doubt, Bouillaud developed an interest in cardiology, and François Magendie (1783-1855), who inspired Bouillaud with an interest in experimental physiology.

In 1818, Bouillaud became an intern in the hospitals of Paris, and in 1823 he received the degree of Doctor of Medicine. After his graduation he became an intern at the Hôpital Cochin under René-Joseph-Hyacinthe Bertin (1757-1828). There he assisted his chief in Bertin's work on diseases of the heart, which was published in 1824.

In 1825, at the early age of twenty-nine, according to Lereboullet, he was named a member of the Académie de Médecine and a year later he received the degree of "agrégé," and became assistant professor.

Bouillaud succeeded Joseph Récamier (1774-1852) in 1831 as professor of clinical medicine of the Faculté de Médecine of Paris. The cholera epidemic occurred during that year and he had occasion to observe many patients suffering from cholera at the Hôpital de la Pitié, where he was a member of the staff. In 1832 he published his observations on cholera and attributed cures to local bleeding combined with cauterization of the spinal column. He also denied the contagious nature of the disease.

Some time later Bouillaud became a member of the staff at La Charité in Paris. In 1835 he published his "Traité clinique des maladies du coeur." In this work is recorded his interesting description of endocarditis, to which he applied the name, and we are presenting to our readers, in translation, his observations on the pathologic aspects of this disease. Trousseau, according to Rolleston, suggested that the term "maladie de Bouillaud" should be given to endocarditis "which was an almost unknown affection until the illustrious professor of the Hôpital de la Charité drew the attention of the medical world to it in a description to which nothing could be added."

Bouillaud also contributed to cardiology his "law of coincidence," which states:[1] "In the great majority of cases of acute generalized febrile articular rheumatism, there exists a variable degree of rheumatism of the fibrous tissue of the heart. This coincidence is the rule, and the non-coincidence the exception."

In the second edition of his treatise on the heart (1841) Bouillaud devoted considerable space to a study of the measurements of the heart, and, according to Rolleston, these were the first accurate methods of weighing and measuring this organ.

Bouillaud also described certain physical signs connected with the cardiovascular system. These included his "bruit de diable," the venous humming sound heard over the internal jugular vein in chlorosis; the "bruit de rappel," the false reduplication of the second sound heard at the apex and characteristic of mitral stenosis; and a tinkling sound sometimes heard on the right side of the apex beat in hypertrophy of the heart. Potain, as we shall later show, credited Bouillaud with being the first to describe gallop rhythm.[2]

Bouillaud is also remembered for his pioneer work in neurology. His most important contribution in this field was his identification of the anterior lobes of the brain as the center of speech. Although he highly esteemed the work of Franz Gall (1758-1828), Bouillaud in 1827 published two papers[3,4] refuting Gall's opinions regarding the function of the cerebellum. Gall thought the cerebellum to be an organ of the instinct of propagation. Bouillaud gave evidence that the cerebellum was the organ of equilibration, station, and progression. He also demonstrated that the lesions of the cerebellum affected co-ordinate movements.

According to Rolleston, Bouillaud is also to be credited with making an advance in the study of acute articular rheumatism, which in France is often referred to as "maladie de Bouillaud."

In 1846, Bouillaud published his last important work: "Traité de Nosographie Médicale." This was an extensive study in five volumes setting forth, in considerable detail, his elaborate doctrines.

Bouillaud was elected a member of the Chambre des Députés for Angoulême in 1840. In 1848, he became dean of the Faculté de Médecine of Paris. In 1862 he was elected president of the Académie de Médecine, and in 1867 he served as president of the first International Medical Congress, which was held in Paris. In 1868, Bouillaud was made a commander of the Legion of Honor.

Bouillaud was at first opposed to the ideas of Pasteur, but later, in 1879, acknowledged the value of his work. He also was extremely critical of the salicylic treatment of rheumatism introduced by Germain Sée in 1877.

Bouillaud's death took place on October 29, 1881, in his eighty-sixth year. Four years later his statue, the work of Verlet, was unveiled at Angoulême in the presence of several of his friends and pupils, including Velpeau, Laboulbène, Cornil, and Potain.

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[1]Rolleston, J. D.: Jean Baptiste Bouillaud, Proc. Roy. Soc. Med. (Sect. Hist. Med.) 24: 1253-1262, 1931.

[2]See page 531.

[3]Bouillaud, J.: Recherches expérimentales tendant à prouver que le cervelet préside aux actes de la station et de la progression, et non à l'instinct de la propagation, Arch. gén. de méd. 15: 64-91, 1827.

[4]Bouillaud, J.: Recherches cliniques tendant à réfuter l'opinion de M. Gall sur les fonctions du cervelet, et à prouver que cet organe préside aux actes de l'équilibration, de la station et de la progression, Arch. gén. de méd. 15: 225-247, 1827.
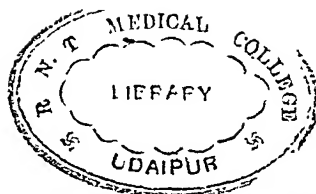
TRAITÉ CLINIQUE

DES

MALADIES DU CŒUR

PRÉCÉDÉ DE

RECHERCHES NOUVELLES SUR L'ANATOMIE
ET LA PHYSIOLOGIE DE CET ORGANE;

PAR J. BOUILLAUD,

PROFESSEUR DE CLINIQUE MÉDICALE A LA FACULTÉ DE MÉDECINE DE PARIS.

—

AVEC DES PLANCHES GRAVÉES.

Scripsi illa, quæ sensuum testimonio
inter labores et tædia iterùm iterùmque
expertus sum.

(AVENBRUGGER.)

TOME PREMIER.

ON THE PATHOLOGY OF ENDOCARDITIS*

By

JEAN-BAPTISTE BOUILLAUD

*EXPOSITION AND APPRECIATION OF THE ANATOMICAL
CHARACTERISTICS OF ENDOCARDITIS*

THE anatomical characteristics of this inflammation, essentially the same as those of pericarditis, offer, however, very remarkable peculiarities depending on the one hand upon the structure of the parts affected by the endocarditis and on the other, upon the presence of blood which circulates incessantly through the cavities lined by this membrane. It is of the greatest importance to know thoroughly the anatomic characteristics proper to each one of the periods of endocarditis, if one would comprehend in a clear and precise manner the principal symptoms which it produces in its course. Thus, in order to have as complete an idea as possible of these characteristics, it is necessary to study successively the alterations of the sero-fibrous tissue itself, the products produced by the inflammatory reaction, and the state of the blood contained in the cavities of the heart. After having thus considered in all their phases the anatomic lesions of which endocarditis may be the source, we shall take care to compare them with the results of inflammations in other organs, and we shall reply to the objections which have been made against our method of deriving the origin of certain organic lesions of the heart.

DESCRIPTION OF THE ANATOMIC ALTERATIONS

I. *First Period of Endocarditis (Period of Sanguinary Congestion, of Softening, and of Ulceration or Suppuration).*

1. The redness of the endocardium is one of the anatomical characteristics the study of which must occupy us in the first place. This redness has been noted in 12 or 13 patients reported earlier. The absence of this redness in one of our patients does not astonish us, since it is known that in cases in which a membranous inflammation leads rapidly to death it may occur that one finds no redness in the cadaver, although this redness may have been present during life. To the innumerable facts of this nature already observed we may add case 5 of our series, in which a marked erysipelatous redness was replaced in the cadaver by a pallor similar to those areas of skin which had not been inflamed.

Nevertheless, in the immense majority of cases, acute endocarditis leaves in its track a redness more or less marked; this color is sometimes rosy and sometimes scarlet, occasionally violet, poppy colored or even brownish. It is partial or general, it often involves only the valves, and almost constantly when it is present in the entire endocardium it is of maximum intensity in the valvular portion of this membrane. Ordinarily, the redness is deeper in the right heart than in the left, which is probably due, at least in part, to the fact that the blood which circulates in the former is less deeply red and less vividly red than that which circulates through the latter. However that may be, this redness is not due to a capillary injection, but to a sort of sanguinary tint of the internal membrane of the heart. Ordinarily it does not penetrate beneath this; it does not disappear on washing, but it does not resist a maceration sufficiently prolonged.

The nature of the redness of the internal membrane of the heart and vessels has been recently the object of numerous discussions. [*Here follows* * *a quotation from Laënnec on this subject.*]

It is difficult to make more researches than I already have on this anatomico-pathologic subject treated here. But, it results from these researches that this redness of the internal membrane of the heart cannot be considered other than as one of the results of the inflammation of this membrane. I am convinced, on the other hand, by a large number of facts, that certain rednesses of the heart and of the vessels are nothing but purely cadaveric imbibition, and I have recognized, with many other observers, that these latter rednesses are almost constant in individuals opened at a period when putrefaction of the body is more or less advanced, above all when the individual has succumbed to an illness accompanied by putrid or typhoid phenomena, cases in which the blood is more liquid than normal, which renders the internal membrane of the vascular system more likely to imbibe.

I do not believe that it is possible to decide by simple inspection, nor by washing or by maceration, whether a given redness of the internal membrane of the heart is the effect of an inflammation or of a cadaveric imbibition. It is necessary to search elsewhere to find the means to solve the grave questions which we examine here. To my way of thinking, one may regard as being of inflammatory nature any redness of the internal membrane of the heart existing in any individual whose body has been opened before any trace of decomposition has been noted, and which during life had presented symptoms which we shall assign in the following article to inflammation of the internal membrane of the heart. But the inflammatory nature of the redness will be more certain if to the conditions which I have already mentioned be added the following: (1) swelling, thickening of the reddened parts, (2) the presence of a certain quantity of pus, pseudomembranous matter or even of adherent dis-

colored clots or fibro-albuminous specks, (3) the coincidence of a similar redness in vessels in which one has positively diagnosed the inflammation before death.

The observations which we have reported in our preceding paragraphs bring together all, or most, conditions under which a redness is to be attributed to inflammation.

A notable thickening of the internal membrane of the heart often accompanies the inflammatory redness above mentioned, when the endocarditis has lasted a certain time; twelve, fifteen, twenty days and more, for example. Nevertheless, this thickening does exist in a marked fashion except over the valves, where the membrane is in a manner double and fortified by fibrous tissue. The fungous swelling of these valves has been reported in many of our cases.

The softening of the internal membrane of the heart is not always evident in this period. It has seemed to me, however, that this membrane breaks with more facility than the normal membrane. At the same time, the surface was a little less polished than the normal, and more or less wrinkled. The cellular tissue beneath the endocardium appeared also, in these cases, to have lost its force of cohesion, and to have become fragile; and because of this to become detached from the endocardium.

One observed sometimes in the acute period of endocarditis erosions of commencing ulcerations of the internal surface of the heart, or of the valves themselves (see Case 50). These ulcerations may become the origin of perforations of the wall of the heart, of the valves, or of the interventricular or intraventricular septa.

2. A purulent or pseudomembranous secretion certainly takes place in endocarditis; but one can imagine that it is often difficult to prove the existence of it. In fact, such is the rapidity and force of the blood flow through the chambers of the heart, that the product secreted little by little by the inflamed endocardium, must be incessantly swept away by this current. Nevertheless, in certain cases a small quantity of true pus or pseudomembranous material is encountered following an acute endocarditis. The pus is sometimes hidden in the centre of a clot or in the net formed by the columnae carnae. The same is true of pseudomembranous material. This latter, of great tenacity, adheres solidly to the parts where it is deposited, and one finds portions of it on the surfaces of the valves, on their free borders, and on their tendons, where it sometimes presents itself under the form of granulations or globules of albuminous consistency or semi-solid fibrin. In certain cases, it is true, one may mistake a simple adherent, discolored, elastic clot for a pseudomembranous production. The error is the more easy to make since pseudomembranous products themselves are formed from solid fibrin, and the error is fundamentally unimportant.

Can an acute endocarditis terminate in gangrene? In reflecting on the extreme rarity of such a termination in membranes which, like the endocardium, are serous, one is naturally inclined to answer this question in the negative. Nevertheless, I have gathered four or five cases in which I have been tempted to believe that it was an internal inflammation of the heart, strongly analogous to certain gangrenous phlegmasias, to which one must attribute the very rapidly fatal termination that took place. The observations made in Cases 22 and 39 appear to me to belong to this category. [*Here the author briefly discusses two cases, one post-traumatic (gas gangrene?) and the other with disordered heart action and high fever which had a reddening of the endocardium at autopsy without ulceration and in one of which bubbles of gas occurred in the very liquid blood in the right heart, which he prefers not to believe was due to post-mortem changes.*]*

Tó return to the subject of gangrenous endocarditis or malignant, as the ancients would have called it, I think that this is a subject to review, and which merits serious researches.

3. We have just passed in review the principal lesions which are presented by an inflamed endocardium, under the double point of view of its structure and its secretory function. Nothing remains for us but to make known the lesions of the blood that is found in the cavities of the heart. But, numerous observations reported in our preceding chapters have indicated that this phlegmasia commonly carries with it the coagulation of a greater or lesser quantity of blood which circulates through the chambers of the heart. In this regard, endocarditis behaves itself in the same way as arteritis and phlebitis. The sanguinary concretions formed under the influence of an acute endocarditis must not be confounded with the ordinary clots found in the heart, especially with those which develop after death. The concretions consecutive to acute endocarditis are white, colorless, elastic, glutinous, adherent to the walls of the heart, wound around the valvular tendons strongly analogous to pseudomembranes; some of these at times present points or red lines, which are nothing but the rudiments of vessels.

The concretions which we have examined differ markedly in respect to volume and configuration. They ordinarily are prolonged into the great vessels. Everything being equal, they are more voluminous, more abundant in the right heart than in the left. Their maximum adherence in general occurs near the free edges of the valves, where one finds fragments after repeated washings. (It is probable that these little fibrinous masses may organize and transform themselves into vegetations or granulations.)

II. *Second Period of Endocarditis (Period of Organization of Secreted Products or of a Portion of Fibrinous Concretions).*

When endocarditis does not terminate in prompt resolution, if it is prolonged a considerable time (fifteen, twenty, thirty days or more), the inflamed tissues are more or less thickened, and the plastic part of the products abnormally secreted by these tissues pass from an amorphous state into a state of organization. Then, according to accidents of position, of configuration or of composition of the organizable matter, one encounters either vegetations or granulations, either cellulo-fibrinous adhesions or fibrinous or serofibrinous layers, and so forth.

The vegetations or granulations have a remarkable predilection for the valves and particularly for their free border. Also, one finds them in certain cases on the internal surface of the cavities of the heart and especially the auricles (Observation XLVII of *Traité de l'auscultation médiate* offers a remarkable example*). They have been divided by M. Laënnec into two divisions: globular vegetations and verrucous vegetations. The first appear to us more exactly described as albuminous or fibrinous granulations. The expression "verrucous" is happily chosen, for the vegetations or excrescences to which they apply resemble warts. One may thus designate them under the denomination of "cornified" or "cartilaginous" vegetations or excrescences.

Do these two kinds of vegetations, granulations or excrescences, represent essentially different entities or are they simply two manifestations of a single accidental production? Do some come from the organization of a portion of solidified fibrin and the others from the organization of a true pseudomembranous exudation? Before considering their mode of generation let us begin by describing them in detail.

The albuminous or fibrinous vegetations (*globuleuses* of M. Laënnec) are soft, easily crushed, like too solid albumin or a half-organized fragment of fibrinous pseudomembrane. They may be detached by a light traction. Their color is grayish white or yellow, mixed sometimes with a rosy tint, or definitely red. These granulations have appeared to me to be analogous to those which are sometimes found on the surface of the chronically inflamed pleura, the pericardium or the peritoneum; the resemblance is even so striking that one would have difficulty in distinguishing them, as I have assured myself by comparing the granulations of the pleura with valvular vegetations, both from the same subject.

The verrucous vegetations, very analogous to venereal warts, are held and implanted so firmly, and are rooted with such tenacity, that they may almost be said to form an identity with the tissue to which they are attached. The tissue of these vegetations is, as it were, cornified; it makes a noise under the instrument which divides it like fibrocartilage.

The number of either albuminous or verrucous vegetations is very variable; the same is true of their size and configuration. Some are no larger than a millet seed; others are the size of flax seed or a small pea.

*See page 362.—F. A. Willius, 1940.

They are sometimes isolated, scattered, discrete, as it were, while others are united in groups, confluent, or so disposed as to imitate cabbage flowers. Their form is often rounded, spherical, but often elongated, cylindrical or flattened. Their surface is sometimes smooth and polished and sometimes unequal and irregular.

It is rare that these vegetations of the valves or of the internal wall of the heart exist alone; usually, as our observations show, they are accompanied by a fibrocartilaginous or calcareous induration of the valves. However this may be, however they are multiplied, confluent, grouped like cabbage flowers, they give rise to a narrowing of the orifices to which are adapted the valves which they overload and whose movements they impede; the existence of this peculiar type of narrowing is an important circumstance to note.

Let us return to the method of production or the pathogenesis of these vegetations. [*Here the author quotes M. Laënnec to the effect that vegetations are probably formed as a consequence of some trouble in the circulation and are polypiform or fibrinous concretions formed through absorption and nutrition analogous to those processes which convert an albuminous false membrane into cellular tissue; also the resemblance between these processes and the crystallizations which form when a thread is placed in a saturated saline solution.*]*†

This comparison seems very ingenious to me and I do not doubt that fibrin organized on the edge of valves may organize into vegetations. But this is no reason to deny the role that endocarditis plays in the production of these vegetations, for as we have already demonstrated by positive facts, this phlegmasia determines the formations of organizable fibrinous concretions. M. Laënnec is thus wrong in opposing our opinion, in saying that "if the inflammation of the internal membrane of the heart was the efficient cause of the vegetations in question, they should have as origin and common ancestor a false membrane extending like a layer over the valves, which does not occur."

But if we admit that fibrin, solidified under the influence of endocarditis or under any other influence, if you will, may be transformed into vegetations, it will be permissible to admit also that pseudomembranous matter, secreted by the inflamed endocardium, may like fibrin itself of which it is really a modification, dispose itself so as to constitute small rounded masses which organize themselves little by little into vegetations. Such a doctrine is the more legitimate, since the vegetations, similar to those developing on the interior surface of the heart, also develop on the serous exterior of the same organ, as also on the surface of the pleura and of the peritoneum in certain cases of pseudomembranous inflammations of these membranes. But, in these cases, one cannot attribute the vegetations observed to polypiform concretions.

But again, in supposing that the vegetations of the internal membrane of the heart always had their origin from a polypiform concretion, we should not be less authorized to place it among the accidental effects of endocarditis since, by the statement of M. Laënnec himself, it is one of the sanguinary concretions due to inflammation. It remains only to determine in which cases the polypiform concretions which may give rise to vegetations should have been really consecutive to a generalized or partial endocarditis. But we have limited ourselves to report here cases of vegetations coinciding with definite traces of an old endocarditis.

The adhesions that endocarditis may leave in its wake have not been mentioned by any of the authors of the *traités sur les maladies du coeur.* I have reported six observations in this work. One can easily conceive why adhesions should be less frequent in endocarditis than in inflammations of other serous membranes. The torrent of blood which flows through the cavities of the heart, the movements of the valves, are powerful obstacles to the formation of these structures of which we speak. Nevertheless, in spite of these obstacles, adhesions sometimes form, and as we might foresee, these adhesions are formed at points where the above mentioned obstacles are at a minimum. Thus, we have encountered them between the less movable leaflets of the valves and on the corresponding wall of the ventricles.

These adhesions are in general very firm. The cases which we have cited show their usual disposition. Such adhesions inevitably cause difficulty in the circulation, since they do not permit the valves completely to close the orifice to which they are adapted.

There is another type of adhesion of the valves which is more common than the preceding, notably that which establishes itself between the opposing borders of the valvular leaflets in certain cases of narrowing of the orifices, which we will describe below.

As a consequence of the adhesions of the endocardium, one must mention the organized false membranes which cover more or less of the surface. There are such which cover nearly the whole area of the cavities of the heart. One may even find many layers organized, one above the other.

Instead of false membranes as extensive as the above, one finds them sometimes hardly more than four, five or six lines in diameter, which I designate under the name of *spots* of the endocardium. They are similar to the milky spots of the pericardium, and also to certain white spots of the cornea, whose name I must give them. One can lift them easily with forceps, underneath one finds the endocardium intact or only a little more opaque than normal.

In a great number of cases, the thickening of the internal membrane of the heart is due to the presence of organized false membranes which we have just mentioned. Also, there are other cases in which the endocardium is

truly thickened, hypertrophied, and then has lost its transparence, and the surface has become less polished, and wrinkled, unequal and a little villous.

The hypertrophic thickening of which we speak extends also to the fibrous tissue as well as to the subendocardium cellular tissue. I have found frequently hypertrophy of the valves and of the tendinous zone where their adherent border is inserted. The bicuspid valve is above all subject to this consecutive hypertrophy, and if there also occurs a slight narrowing of the orifice, one sees on the side of the auricle a wrinkling of the adherent border of the valve, which we will describe later.

III. *Third Period (Period of Cartilaginous, Osseous or Calcareous Induration of the Endocardium in General, and of the Valves in Particular, With or Without Narrowing of the Orifices of the Heart).*

We have seen in the preceding chapters the internal membrane of the heart and valves thicken and hypertrophy and the products secreted through the influence of the inflammation organize into cellulo-fibrous, fibrous or even fibro-cartilaginous tissue. (The tissue of verrucous vegetations belongs to the latter category.) It remains to us to study the cartilaginous, osseous or calcareous productions which remain as a consequence of an endocarditis.

The productions considered here present themselves under diverse forms. Sometimes they are simple circumscribed points of the size of a lentil, sometimes sheets or plaques of the size of a fingernail or a piece of money of 10 *sous* or larger; sometimes more or less rounded masses. The entire valves are sometimes converted into cartilaginous or osseous tissue. Also, the fibrous zone of the orifices of the heart and the points of the valves are the parts affected by preference by cartilaginous degeneration. Between the plaques or incrustations of the valves, one finds spaces in which the tissue is normal or presents only a slight degree of hypertrophy.

The osseous or calcareous incrustations of the valves have the most varied configuration. Some are bent in an arc, or in a hoop; others are elevated and elongated in the form of stalactites, of pyramidal or irregular form; still others are rounded into spheres and resemble true calcareous concretions of which the volume is sometimes that of a pigeon's egg or even of a small hen's egg. These kinds of calculi or stones developing on the surface or in the substance of the valves, are ordinarily covered with sharp points, or inequalities on their surfaces, and simulate the calculi designated as *muraux* (mural calculi). These ossifications or petrifactions sometimes send prolongations into the substance of the heart.

The chondrified, ossified or petrified valves show the most varied changes in their conformation. When the accidental productions of which they are the seat consist of simple points or small blades, the thickened and slightly rigid valves conserve still their mobility, and can rise and fall as in the normal state, but when the organized valves are more profoundly altered,

when the degeneration has invaded them in all of their parts, they become more or less completely unable to perform the functions that nature has confided to them. In certain cases their leaflets are folded or rolled upon themselves, and then they represent straight ribbons; they then are too short and too immobile to be able to close the orifice to which they are adapted. The *insufficiency* of the valves is evident in this kind of deformation; the orifice may be dilated, which renders their insufficiency greater. The valvular leaflets may be perforated or torn, at the same time that they are indurated and thickened; in one case one of the valves of the aorta, almost entirely detached, hung and floated, so to speak, in the cavity of the aorta.

In a great number of cases, the valves affected with induration and thickening are united and attached by their neighboring edges, and from a sort of membrane or diaphragm pierced in its center by a narrow opening, sometimes rounded, sometimes oval or elliptic. The thickening and induration sometimes invade the chordae tendinae and columnae carneae.

The large number of observations that we have reported give an idea of the principal variations in form, extent and aspect that the productions that we are studying may produce.

The narrowing of the orifices of the heart consecutive to the different kinds of indurations and valvular transformations merits a detailed description. In giving this description we shall have occasion to designate a few other dispositions of the valves which must be studied with the narrowing itself.

The narrowing of the orifices of the heart, following different kinds of thickening and induration of the valves, is the gravest anatomic accident that endocarditis may carry in its wake. In order to appreciate this, one must recall the normal dimensions of the orifices of the heart, as they have been established in our *Prolegomenons*.

The degrees of narrowing of the orifices of the heart are very variable. In extreme degrees, one can hardly introduce the tip of the little fingers, or even the tip of a writing pen, into the narrowed orifice.

The opening which is left between the thick, indurated valves or those valves united by their neighboring borders, is permanent, or constantly open. It is sometimes rounded, oval or elliptic. It resembles in many cases a buttonhole or a glottis of which the lips are represented by the rounded borders of the thickened valve leaflets. This comparison applies particularly to certain narrowings of the left auriculo-ventricular orifice. In a few cases, the leaflets of the bicuspid valve have acquired an enormous thickening and protruded from the side of the auricle; then the retracted orifice may be compared to the orifice of the cervix uteri, and like this imitates a fish mouth. Seen from the auricular side, the circumference of the retracted auriculo-ventricular orifice presents a very pronounced folding, as if this circumference had been folded upon itself;

this disposition gives it the appearance of the external circumference of the anus or the opening of a purse drawn together with cords.

In general, in cases with a considerable narrowing of the orifices, the valves, rendered immobile by their rigidity, are elevated so as to cross the axis of the orifices almost at a right angle. Also, they are often enough reversed in the direction of the blood flow. Then the orifice represents a sort of infundibuliform canal of which the summit presents to the ventricles, if the auriculo-ventricular orifices are the site of narrowing, and toward the aorta and pulmonary artery, if the narrowing affects the aortic or pulmonary orifices. Nevertheless, as for the aortic valves, one observes an inverse disposition, that is to say, they are dejected toward the ventricular cavity.

To give an exact idea of the principal forms that the narrowed orifices of the heart may show, and to emphasize the peculiarities which distinguish the narrowing of each one of these orifices, we have believed it wise to reproduce here, in a note, the description of the principal cases of this kind of lesion, which we have reported in the first section of this chapter. [*A note gives the pathologic description of hearts extracted from case reports by the author, reported in the first part of the book.*]*

We will not terminate this article without remarking to the reader that this narrowing which is so common in the orifices of the heart, as a consequence of a prolonged chronic endocarditis, is a new fact of resemblance between this phlegmasia and those which affect other hollow organs. What physician is ignorant that the urethra, the neck of the bladder, the arteries, the excretory canals for the tears, saliva, bile, the different regions of the digestive tube, and especially the cardia, the pylorus, the junction of the ileum with the cecum, the inferior portion of the rectum etc., etc.; what physician, I say, is ignorant that all these parts just mentioned may undergo a narrowing of greater or lesser degree, as a result of a long and slow inflammation? It is not possible here to mention the results which follow these mechanical lesions, the gravity of which is proportionate to the importance of the function to which they impose a more or less invincible obstacle.

However that may be, the preceding details are sufficient to bring clearly to light this truth to which we have called the attention of the reader: in treating the diseases of the heart in general, that the diverse lesions of that organ engender reciprocally one another; and that lesions of nutrition or of organic or vital action, for example, always result, when they are not arrested or strangled in their cradle, so to speak, by producing physical or mechanical lesions which are too often resistant to all measures known to our art.

1846

WILLIAM STOKES

DESCRIPTION OF HEART BLOCK

WILLIAM STOKES

(Courtesy Medical Classics.)
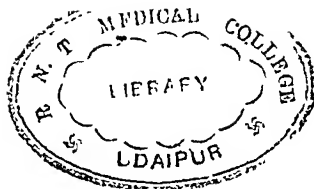
WILLIAM STOKES

(1804-1878)

"We have to do with something which cannot be measured or weighed; something, too, in which experiment can only be used within narrow bounds; an element whose nature is yet unknown, fleeting in its action, and every day producing new combinations, not merely new because they were never observed before, but really new as appearing for the first time."

—William Stokes on medical education.

$\mathcal{W}$ILLIAM STOKES, the second son of Whitley Stokes, was born in Dublin. His distinguished father was a physician and Regius professor of medicine in the University of Dublin, and in 1800 was appointed professor of medicine in the Royal College of Surgeons at Dublin.

William Stokes was tutored by the well-known scholar, John Walker, who taught him the classics and mathematics. Some time later, Stokes studied clinical medicine in Meath Hospital. He learned the auxiliary sciences at both Trinity College and the Royal College of Surgeons at Dublin. He also spent two years in Glasgow, where he worked in chemistry under the direction of Professor Thompson. From Glasgow, Stokes went to Edinburgh, where he completed the required studies for the medical degree.

At Edinburgh it was Stokes's good fortune to be the pupil of William Alison, professor of medicine. And it was the stimulation received from Alison which led Stokes at an early date to achieve prominence among the pioneers of medical science.

At about this time the medical profession was much concerned with diagnosis of diseases of the chest, a concern that was brought about by the delayed acceptance of Auenbrugger's system of percussion and the development of auscultation by Laënnec. William Stokes, shortly before he was graduated from medical school, published the first systematic work in the English language on the use of the stethoscope,[1] "An Introduction to the Use of the Stethoscope," printed in Edinburgh in 1825 by Machlachlan and Stewart. This work and two subsequent lectures,[2] published in 1828, formed the basis for his more mature account of diseases of the chest which he published in 1837.

After Stokes had been graduated from Edinburgh (1825), he returned to Dublin to become physician to the Dublin General Dispensary. In 1826, at the age of twenty-two, he succeeded his father as physician to the Meath Hospital. At Meath he had as a colleague the distinguished Robert Graves, who became his lifelong friend. Stokes and Graves did much at Meath to improve the system of clinical teaching.

In April, 1828, Stokes was married to Miss Mary Black, to whom he had been engaged for three years.

Following Graves's prediction of the event in 1826, Asiatic cholera broke out in Ireland at some time in 1832. Stokes and Runley reported the first case of cholera

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[1] Dr. Cullen, to whom Stokes had dedicated his work, and Sir J. Forbes had earlier published reports of cases illustrating the practical use of the stethoscope.

[2] *Two Lectures on the Application of the Stethoscope to the Diagnosis and Treatment of Thoracic Disease*, Dublin, 1828, Hodges and McArthur.

in the Dublin epidemic, an achievement which, as it turned out, entailed considerable personal risk. The two physicians had been sent to inquire into the cause of a certain mysterious death which had occurred at Kingstown. After their inspection, they pronounced the deceased person to have died of Asiatic cholera of the worst type. The crowd which had gathered outside the house of the deceased person received the announcement calmly at first, then, realizing the horror of the situation, invoked its wrath on the physicians. Members of the mob hurled stones and sticks at them. Stokes and Runley escaped in their carriage, but it was well battered when they reached home.

Stokes's first work of major importance was his treatise on diseases of the chest,[3] published, as we have mentioned, in 1837. This book did much to elucidate the phenomena of thoracic disease. In his book Stokes followed the line of investigation initiated by Corvisart and Laënnec. According to Corrigan[4] Stokes's volume added much to the work of Laënnec. Stokes, moreover, clarified an issue which the followers of Laënnec had neglected to elucidate: Stokes pointed out that in diagnosis, physical signs must be associated with symptoms.

Among the important observations found in Stokes's book were: (1) the discovery of a stage of pneumonia prior to that described by Laënnec as the first stage, (2) the discovery of a displacement of the heart as the result of the rapid absorption of pleuritic effusion in the right side, and (3) the employment of the stethoscope as an aid to the detection of foreign bodies in the air passages.

After the publication of Stokes's work on thoracic disease, he received many honors. The University of Dublin granted him the degree of Medicinae Doctor, honoris causa; he was elected a fellow of the King's and Queen's College of Physicians in Ireland; he was made an honorary member of the Imperial Academy of Medicine of Vienna, and of the royal medical societies of Berlin, Leipzic, Edinburgh, and Ghent. His fame had even spread to the United States, where he was elected to the National Institute of Philadelphia.

Such a favorable reception of his work provided a healthy stimulus to Stokes, both in his writing and in his clinical teaching. During the next decade his attention was drawn chiefly to the diseases of the heart and he contributed many papers on this subject to the Dublin "Quarterly Journal of Medical Science." He was an editor of this journal, in association with Robert Graves and William Parker, from 1836 until 1842. The articles he contributed to it formed the basis for another work equally important to his earlier work on thoracic diseases. This was "The Diseases of the Heart and the Aorta," published in 1854 at Dublin by Hodges and Smith.

From the chapters of the aforementioned work we have reprinted his classic description of "Cheyne-Stokes respiration" in connection with fatty degeneration of the heart.[5] This type of respiration had been noted earlier by John Cheyne, but Cheyne did not associate any diagnostic importance with the syndrome. Stokes also referred in his book on the heart to his memoir on slow pulse.[6] Therein he had described the condition first noted by Adams which is now called "heart block with the Adams-Stokes syndrome." This passage, also, we are reprinting.

Stokes in his book on the heart also advocated pursuance of a system of graduated muscular exercises to aid in the removal of cardiac debility, especially among younger persons. The book is additionally famous for its accurate descriptions of pericarditis, valvular diseases, and weakening of the heart in typhus fever.

[3]A Treatise on the Diagnosis and Treatment of Diseases of the Chest, Part I, Diseases of the Lung and Windpipe (no more published). Dublin, 1837, Hodges and Smith.
[4]Quoted by Stokes's son in William Stokes, His Life and Work (1804-1878), London, 1898, T. F. Unwin, p. 65.
[5]See pp. 484-489.
[6]This was first published in the Dublin Quarterly Journal of Medical Science 2: 73-85, 1846.

In 1861, the honorary degree, "Legum Doctor," was conferred upon Stokes by the University of Edinburgh. In 1865 the University of Oxford conferred on him the same degree. Stokes was further honored in 1867 by election to the presidency of the British Medical Association. The annual meeting of the association was held that year in Dublin. Through Stokes's efforts, graduate education in state medicine was established at Dublin. Oxford and Cambridge soon followed in this respect.

Stokes was elected to the presidency of the Royal Irish Academy in 1874. Failing health did not permit him to hold this office for more than two years. In that same year (1874) Stokes received the honorary degree, "Legum Doctor," from the University of Cambridge. During this time Foley was working on a statue of Stokes. It was unveiled in 1876 and now stands in the hall of the Royal College of Physicians in Dublin.

In 1876 Stokes was presented with the Prussian order, "Pour la mérite," originated by Frederick the Great. He was one of the few physicians ever to receive this honor. On a professional visit that same year he was injured by a fall from a car. This accident was followed by symptoms of spinal concussion, and it seemed to be the cause of the development of the paralytic affliction which gradually weakened him and finally deprived him of the use of his limbs. Early in November, 1877, he suffered a sudden paralytic seizure from which he never rallied. On January 6, 1878, he quietly passed away.
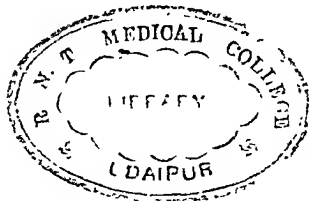
OBSERVATIONS ON SOME CASES OF PERMANENTLY SLOW PULSE*†

By

WILLIAM STOKES, M.D.

Physician to the Meath Hospital

IN THE fourth volume of the Dublin Hospital Reports, Mr. Adams has recorded a case of permanently slow pulse, in which the patient suffered from repeated cerebral attacks of an apoplectic nature, though not followed by paralysis. The attention of subsequent writers on diseases of the heart, has not been sufficiently directed to this case, which is an example of a very curious and, as there is reason to believe, special combination of symptoms. The following cases will still further elucidate a subject on which there is but little information extant:—

Case I. Repeated pseudo-apoplectic attacks, not followed by paralysis; slow pulse, with valvular murmur.

Edmund Butler, aged sixty-eight, was admitted into the Meath Hospital, Feb. 9th, 1846. He stated that his health had been robust, until about three years ago, at which time he was suddenly seized with a fainting fit, in which he would have fallen if he had not been supported. This occurred several times during the day, and always left him without any unpleasant effects. Since that time he has never been free from these attacks for any considerable length of time, and has had, at least, fifty such seizures. The fits are very uncertain as to the period of their invasion, and very irregular as to their intensity, some being much milder and of shorter duration than others. They are induced by any circumstance tending to impede or oppress the heart's action, such as sudden exertion, distended stomach, or constipated bowels. There is little warning given of the approaching attack. He feels, he says, a lump first in the stomach, which passes up through the right side of the neck into the head, where it seems to explode and pass away with a loud noise resembling thunder, by which he is stupified. This is often accompanied by a fluttering sensation about the heart. He never was convulsed or frothed at the mouth during the fit, but has occasionally injured his tongue. The duration of the attack is seldom more than four or five minutes, but sometimes less; but during that time is perfectly insensible. He never suffered unpleasant effects after the fits, nor had anything like

paralysis. His last fit occurred about one month before admission. He has never heard it remarked that there was anything peculiar about his heart or pulse. At first he found that spirits was the best restorative or prophylactic, but lately he has not used them, being "afraid to die with spirits in his belly."

On admission, he was haggard and emaciated, but seemed the wreck of what was once a fine, robust man. He lay generally in a half drowsy state, but when spoken to was perfectly lively and intelligent.

What he sought admission into hospital for was an injury he had sus-stained, by a fall, on the left shoulder, this, however, was of no conse-quence, and he soon recovered under the use of an anodyne liniment.

He makes no complaint of his general health; his appetite is good, and he sleeps well; bowels regular, and, in fact, all the functions are in good order. He has, however, some cough, attended with a slight mucous ex-pectoration. His intellectual powers are perfect. He complains of a feel-ing of chilliness over the body, and is never warm except when close to the fire. This has long been the case; and he says that each day he gets a periodical chill, generally in the afternoon, which is followed by increased heat of the surface, but without sweating.

On percussion, the chest is universally resonant. The respiratory mur-mur loud, and combined, more especially posteriorly, with large mucous râles. The impulse of the heart is extremely slow, and of a dull, pro-longed, heaving character, giving the idea of feeble as well as of slow action. The first sound is accompanied by a soft bruit de soufflet, which is prolonged until the commencement of the second sound, and is heard very distinctly up along the sternum, and even into the carotid arteries. The second sound is also imperfect, though very slightly so; the imperfection being much more evident after some beats than after others. Pulse twenty-eight in the minute, of a prolonged, sluggish character; the ar-teries pulsate visibly all over the body, but no bruit is audible in them. They appear to be in a state of permanent distention; the temporal ar-teries ramifying under the scalp, just as they are seen in a well-injected subject. All the other cavities and viscera appear to be in a perfectly healthy state. Urine, neither acid nor alkaline; of a high colour, clear; specific gravity 1.010; and does not afford a precipitate with nitric acid. He was ordered four ounces of wine, and a liniment for the shoulder.

February 17th. The pulse has varied from twenty-eight to thirty in the minute. The cardiac murmurs continue unchanged; that with the first sound is plainly audible over the upper part of the thorax, but most evident along the course of the aorta.

21st. Pulse thirty. Cough quite gone. Has been complaining of a feeling of the "lump in the stomach" for several days, and was once threatened with the approach of a fit during the night; it passed off, how-ever, without becoming a true attack.

23rd. An edematous swelling has appeared behind the left ear, extending up the side of the head, slightly tender on pressure; no redness; has had no shiverings, tongue clean; bowels free. Pulse up to 36.

March 3rd. On the 24th of February the edema had left the left side, and made its appearance on the right, from which it was dispersed on the following day by the application of poultices. The pulse fell to the usual range.

His aspect and general health are greatly improved since his admission. He gets up every day, and is much stronger. The shoulder is almost quite well. The pulse has continued at about 28 or 30. He says he has had two threatenings of fits since his admission, both occurring in bed, and both warded off by a peculiar manoeuvre; as soon as he perceives symptoms of the approaching attack, he directly turns on his hands and knees, keeping his head low, and by this means, he says, he often averts what otherwise would end in an attack.

4th. He has mentioned, for the first time today, that he is much troubled with irritability of the bladder, so that he is obliged to rise very often during the night to pass water. His urine was examined and found to be healthy. Specific gravity 1.015. He has been subject to this for the last twelve months, and it probably depends on the disease of the prostate so common in old men.

We remarked today, that on listening attentively to the heart's action, we perceived that there were occasional semi-beats between the regular contractions, very weak, unattended with impulse, and corresponding to a similar state of the pulse, which thus probably amounts to about 36 in the minute, the evident beats being only 28, so that there must be about eight of these semi-beats in the minute; but these signs are very indistinct.

14th. Health improving; has had no fit; no cough. Both morbid sounds are loudest over the sigmoid valves, and thence along the aorta. No semi-beats audible. Pulse 29; not quite so prolonged as before.

18th. He complains today of palpitation, and a feeling of uneasiness about the heart;—the impulse is increased and is found to consist of two distinct pulsations. The bruit, with the first sound, is somewhat louder than before. On listening attentively, there are heard occasional abortive attempts at a contraction, probably about four in the minute. They do not destroy the regular intervals between the stronger sounds, but are heard, as it were, filling up the interval. We could not recognize a corresponding state of the pulse, which counted 32 in the minute.

After this, little change was observed. His health continued improved; he had no fit, or threatening of one; and he appeared anxious to leave hospital, in order to go to work again. The pulse continued about the same standard, and regular; I believe it never exceeded 36 in the minute

since his admission into the hospital. The physical signs remained unchanged, as was observed the day before he left the hospital. An examination of the lungs revealed no morbid sign, the bronchial râles, heard at the time of admission, having quite disappeared.

He left the hospital in March, intending to go for some time into the country before he resumed work. He was advised to be careful not to over-exert himself; and never to allow himself to be bled when threatened with one of his fits.

Within the present month (June) this patient has been again admitted into hospital. The cardiac phenomena remain as before, but a new symptom has appeared, namely, a very remarkable pulsation in the right jugular vein. This is most evident when the patient is lying down. The number of the reflex pulsations is difficult to be established, but they are more than double the number of the manifest ventricular contractions. About every third pulsation is very strong and sudden, and may be seen at a distance; the remaining waves are much less distinct, and some very minor ones can also be perceived. These may possibly correspond with those imperfect contractions which have been already noticed in the heart. The appearance of this patient's neck is very singular, and the pulsation of the veins is of a kind which we have never before witnessed.

He has had scarcely any of the cardiac attacks since he was discharged; he refers the premonitory sensations to the right supra-clavicular region, but states that he has often experienced them without any loss of consciousness following.

The next case exhibits a similar condition of the heart, but the pseudo-apoplectic attacks did not occur.

Case II. Anemic condition; very slow pulse, with valvular murmur; death, apparently from syncope.

A man, upwards of fifty years of age, was admitted, presenting much of the general characteristics of senile phthisis. His skin was of a pale yellowish tint and his whole appearance indicated great debility. He complained of cough and dyspnea, but did not refer any of his suffering to the region of the heart. His pulse was generally 35 in the minute, though occasionally rising to 40. The action of the heart was regular, but feeble, and a valvular murmur with the first sound, precisely similar to that in mitral-valve regurgitation, was always audible. This became louder on ascending the sternum, and was most intense on the right side, at the anterior articulation of the second rib. We were inclined to consider this as an example of mitral valve disease, and supposed at first that the aortic murmur might result from anemia. The patient died without any struggle. On dissection, the mitral valve was found healthy. The aortic valve was thickened and narrowed, but not permanently patent. Water poured into the aorta did not pass into the ventricle; the heart was soft and flabby, and though not an example of complete fatty degeneration,

was covered by a very thick layer of fat. The aorta presented several atheromatous patches.

In this case the second sound remained normal; there was no regurgitation into the ventricle. The valve was sufficiently diseased to cause a murmur with the first sound, but from its power of closing completely, the second remained unaltered.

The co-existence of aortic murmur with the symptoms of weakened heart in both these cases is important, for it should appear that this combination is one of frequent occurrence, we shall have less difficulty in recognizing an obscure disease of the heart. There is no reason to believe that there is any necessary connection between the weakened, or fatty state of the heart, and disease of the aorta or its valves; but that the combination is frequent appears probable from the following considerations:

First.—In the two cases which have now been given, we see the combination of slow pulse with aortic murmurs.

Secondly.—In one of these, organic disease of the aorta was found on dissection.

Thirdly.—In Mr. Adams' case the aortic valves were studded with specks of bone. . . . The state of the aorta is not noticed: but the carotids and middle arteries of the dura mater presented bony depositions.*

Fourthly.—In a case published by Dr. Cheyne, in the second volume of the *Dublin Hospital Reports*, in which the heart had greatly degenerated into fat, the valves were sound, but the aorta was studded with atheromatous concretions.

Fifthly.—Professor Law, in his original and important observations on the connection between disease of the heart and brain, in the seventeenth volume of the *Dublin Journal of Medical Science*, gives an account of the appearances observed in examining the body of the Earl of K., and states that the pulse was remarkably infrequent, sometimes not exceeding twenty-five beats in the minute. The patient was subject to syncope. The examination was made in London, and no mention is made of the state of the muscular substance of the heart; but it was found that the semilunar valves of the aorta were thickened and partially ossified, so that they could not effectually have closed the orifice. The brain was extensively softened, and the ventricles distended with a limpid fluid, and the substance of the left hemisphere, both cortical and medullary, was so softened as to present an almost creamy consistence. The arteries at the base of the brain presented opaque yellow depositions.

This case was, in all probability, an example, if not of fatty degeneration, at least, of a weakened state of the ventricle. It is another example of the combination of a singularly slow pulse, tendency to syncope, and disease of the aortic valve.

*See account of Robert Adams, page 398.

I am indebted to Mr. Adams for the particulars of an interesting case of slow pulse, with lesion of the aortic orifice, and remarkable softening of the left ventricle. The patient had been in excellent health up to within a few months previous to his death. He had no palpitation, dyspnœa, nor irregularity of the pulse. He had been exposed to various debilitating causes, and, when seen by Mr. Adams, presented a slow pulse and visible pulsation of the arteries of the neck. The pulse fell to below forty, and a loud bruit de soufflet could be heard along the aorta and in the region of the heart. Mr. Adams found the heart to be one of the most friable he had ever met with, breaking down under the slightest pressure of the fingers. The valves of the aorta were less diseased than could have been expected, considering the state of the pulse, and the visible pulsations noticed in all the arteries. The valves were not inadequate to perform their functions, from their being diseased or altered in their structure of form; but the calibre or area of the aorta was so expanded that they could not prevent reflux into the ventricle.

I have lately seen another case presenting the combination of a pulse under thirty, repeated pseudo-apoplectic attacks, not followed by paralysis, and distinct valvular murmur with the first sound. The gentleman is advanced in life, but enjoys very good general health. He has always found that the attacks were increased whenever he was lowered by regimen or medicine. He takes a moderate quantity of wine, and is thus able to ward off the malady.

The preceding observations go to prove that the combination of the permanently slow pulse, with a diseased condition of the aortic opening, is not uncommon. We owe to Dr. Corrigan, the important practical observation, that in cases of permanent patency of the aortic valve, the patients do not generally bear a reducing system, but are best treated by a tonic, or even stimulating regimen; and I entirely agree with Professor Law in his opinion, that the pseudo-apoplectic attacks, in cases of slow pulse and weakened left ventricle, are more frequently attributable to a diminished or feeble circulation, than to one of active congestion.

We have thus seven cases of permanently slow pulse. In five, organic disease of the aorta or the valves, or both, was discovered on dissection; and in four, a manifest aortic murmur existed; in two of the cases the second sound was normal; and in two there was the murmur of regurgitation in the aortic valve.*

I do not believe, however, that the aortic murmur is any direct sign or necessary combination of the weakened heart. Its occurrence in these cases manifestly arises from the combination of aortic disease; and we have abundant evidence that a weakened heart, without aortic disease,

*In Dr. Robert Smith's published cases of fatty degeneration of the heart, the valves were healthy. The patients were both very old women, and no stethoscopic observation is recorded. The pulse was very slow. These patients were not under Dr. Smith's care. See also the important case of fatty degeneration of the heart, communicated to the Pathological Society by Mr. Carmichael—Transactions of the Society for 1840.

may exist, and yet no murmur be produced. In the typhoid softening of the heart, we have rarely recognized a valvular murmur; and where it did occur, there was reason to believe that carditis had supervened. The typhoid softening, with a pulse from 30 to 40, commonly exists without any murmur.

In Dr. Cheyne's patient a remarkable state of the respiration was observed for some time before death. "For several days," says Dr. Cheyne, "his breathing was irregular; it would entirely cease for a quarter of a minute, then it would become perceptible, though very low, then by degrees it became heaving and quick, and then it would gradually cease again. This revolution in the state of his breathing occupied about a minute, during which there were about thirty acts of respiration."

I once witnessed this condition of breathing, but had not an opportunity of making a dissection. The patient was a gentleman of about sixty years of age, and of spare habit; his ailments commenced with a sudden and severe attack of dyspnea, which subsided, leaving him to all appearances perfectly well; this returned at irregular intervals. When I saw him he had a full, soft, pulse, and a loud though varying murmur with the first sound, propagated into the aorta. At the top of the sternum the murmur was intense, and on several occasions the arteries seemed to pulsate with a force much greater than could be expected from the impulse of the heart. For more than two months before his death, this singular character of respiration was always present, and so long would the periods of suspension be, that his attendants were frequently in doubt whether he was not actually dead. Then a very feeble, indeed barely perceptible inspiration would take place, followed by another somewhat stronger, until at length high heaving, and even violent breathing was established, which would then subside till the next period of suspension. This was frequently a quarter of a minute in duration. I have little doubt that this was a case of weakened and probably fatty heart, with disease of the aorta.

Professor Law has lately communicated to me the following note of a case under his care in Sir Patrick Dun's Hospital. It is an example of a very weak heart, with the pseudo-apoplectic attacks.

Colin Baird, admitted March 10th, 1846, aetat. 44, states that about three years ago he was suddenly attacked with a fit, which he describes as coming on without any warning; his sight suddenly failed, and he fell down; this fit only continued a minute or so, leaving him stupid for some time. After the first attack the patient states that his health was bad for two months, when he resumed his trade, being occasionally attacked at irregular intervals, varying from two to three months, till the attacks became more frequent; and at length came on ten or twelve times in the twenty-four hours. These attacks are induced by smoking, or drinking

WILLIAM SENHOUSE KIRKES

(Courtesy St. Bartholomew's Hospital Journal.)

WILLIAM SENHOUSE KIRKES

(1823-1864)

WILLIAM SENHOUSE KIRKES, according to Power, was born at Hilker, in North Lancashire, England, in 1823. He received his primary education at the Cartmel Grammar School. He then became apprenticed to a firm of surgeons at Lancaster. In 1841, Kirkes began his studies at St. Bartholomew's Hospital, where he proved to be a brilliant student. In 1812 he received the highest grades in chemistry and in 1843, he ranked first in surgery. In 1844, he was first in medicine, midwifery, medical jurisprudence, and clinical medicine.

In 1846, Kirkes received his degree of Doctor of Medicine from the University of Berlin. St. Bartholomew's Hospital appointed him medical registrar and demonstrator of morbid anatomy in 1848. In 1850 he became a licentiate of the Royal College of Physicians, and in 1855 he was elected a fellow of that organization. In 1856 he delivered the Goulstonian Lecture.

Kirkes was appointed assistant physician to St. Bartholomew's Hospital in 1854, and physician in 1864, a few months before his untimely death. He lectured at St. Bartholomew's Hospital on botany, and, with Dr. Patrick Black (1813-1879), delivered joint lectures on medicine.

The diseases of the vascular system were of special interest to Kirkes, and in 1852, he contributed the first English article on "Embolism from Intracardiac Coagula," in which he confirmed Virchow's views, which had been published a few months earlier. Because Kirkes's description of the condition leaves little to be desired, we have included it among our classic accounts.

Kirkes expected to write a work on diseases of the heart, but unfortunately, death intervened before he had collected all his material.

Dr. Kirkes was a member of the commission appointed by the Admiralty and Horse Guards to make an investigation of the problem of venereal disease. He had served in this capacity for a short while, when suddenly he was afflicted by pneumonia accompanied by pleurisy and pericarditis. The disease made rapid progress and he died on December 8, 1864.

Kirkes made a transcription of Sir James Paget's "Lectures on Physiology" which was published in 1848 as the "Handbook of Physiology, by W. S. Kirkes assisted by James Paget." Paget's name was omitted from subsequent editions of this work, which was published as "Kirkes' Physiology" until the issuance of the fourteenth edition, at which time Halliburton became the author.

The friends and former students of Kirkes raised a fund of money by subscription to provide a memorial to him. For many years this consisted of a gold medal awarded annually to the student of St. Bartholomew's Hospital who passed the best examination in the diagnosis and treatment of patients in the medical service in the wards of the hospital. In 1885, Mrs. Kirkes provided a fund that caused the aggregate of the annual prize to be thirty pounds, in addition to the medal.

ON SOME OF THE PRINCIPAL EFFECTS RESULTING FROM THE DETACHMENT OF FIBRINOUS DEPOSITS FROM THE INTERIOR OF THE HEART, AND THEIR MIXTURE WITH THE CIRCULATING BLOOD*

By

WILLIAM SENHOUSE KIRKES, M.D.

Licentiate of the Royal College of Physicians, Registrar and Demonstrator of Morbid Anatomy at St. Bartholomew's Hospital

THAT the fibrinous principle of the blood may, under certain circumstances separate from the circulating fluid during life, and be deposited within the vascular system, especially on the valves of the heart, is a fact so clearly established and so generally admitted, that I need only, at the outset of the communication I have the honour to present to this Society, allude to it as a settled truth, and refer, for the proofs, to the various general works on diseases of the heart and blood-vessels, and to such special essays on the subject as those of Dr. Burrows[1] and Dr. Hughes.[2] From these sources may also be gathered nearly all that is yet known respecting the various conditions under which the deposition of fibrine takes place, and the several forms which the deposits assume. Into these general details I do not propose entering, my object being simply to consider the effects which the deposits may produce on the system at large. It may, however, be premised that the forms of fibrinous concretions to which my observations chiefly apply, are, first, the masses usually described as Laënnec's globular excrescences; and, secondly, the granular or warty growths adhering to the valves and presenting innumerable varieties from mere granules to large irregular fungous or cauliflower excrescences projecting into the cavities of the heart.

Avoiding all discussion concerning the origin of these latter growths, I proceed at once to state that in whatever way they may originate, they are, when once formed, full of peril, and often remain so even long after the circumstances which gave rise to them have passed by. If of large size and only loosely-adherent, as they often are, one or more masses of even considerable magnitude may at any time be detached from the

valves and conveyed with the circulating blood until arrested within some arterial canal which may be completely plugged up by it, and thus the supply of blood to an important part be suddenly cut off, and serious, even fatal results ensue. Or, the deposits on the valves may be detached in smaller masses, and pass on into arteries of much less size, or even into the capillaries, where, being arrested, they may cause congestion, followed by stagnation and coagulation of the blood, with all the subsequent changes which blood coagulated within the living body is liable to undergo. In this way are probably induced many singular morbid appearances often observed in internal organs, and rarely well accounted for. Again, the masses of fibrine may soften, break up, and discharge the finely granular material resulting from their disintegration; and this, mingling and circulating with the blood, may give rise to various disturbances indicative of a contaminated state of this fluid, producing symptoms very similar to those observed in phlebitis, typhus, and other analogous blood-diseases. In one or more of these several ways, and probably in others not yet clearly recognised, fibrinous material detached from the valves, or any other part of the interior of the heart, may be the cause of serious secondary mischief in the body.

It appears unnecessary to insist here on the possibility of any of the various forms of fibrinous deposit found within the heart being detached either spontaneously or by the mere force with which the current of blood passes over the surfaces on which they are placed. For it is well known that after death a very gentle force, sometimes even the slightest touch, will loosen and dislodge both small granular particles and masses of considerable size from the valves and inner surface of the heart. Not infrequently, indeed, lumps of old laminated fibrine of even considerable magnitude are found loose in the cavities of the heart, having probably dropped off before death; and sometimes a mass of this kind may be found some distance along the aorta or pulmonary artery.

It is clear, then, that such fibrinous deposits may admit of being very readily detached, and it must be equally clear that once floating freely in the blood they are exposed to the almost certain consequence of being transmitted with this fluid, and stopped at the first vessel too narrow to allow of their transit.

The parts of the vascular system within which these transmitted masses of fibrine may be found will of course depend, in great measure, upon whether they proceeded from the right or left side of the heart. Thus if they have been detached from either the aortic or mitral valves, they will pass into the blood propelled by the left ventricle into the aorta and its subdivisions, and may be arrested in any of the systemic arteries or their ramifications in the various organs, especially those which, like the brain, spleen, and kidneys, receive large supplies of blood directly from the left side of the heart.

If, on the other hand, the fibrinous masses are derived from the pulmonary or tricuspid valves, the pulmonary artery and its subdivisions within the lungs will necessarily become the primary if not the exclusive seat of their subsequent deposition. A division of the subject being thus naturally formed, I propose to embody the remarks I am about to submit to the Society under two principal heads, considering—

1st. The remote effects resulting from the separation of fibrinous or analogous deposits from the valves or interior of the left side of the heart; and

2d. The corresponding effects produced by the detachment of like deposits from the valves or interior of the right side of the heart.

PART I

ON THE EFFECTS WHICH MAY RESULT FROM THE SEPARATION OF FIBRINOUS DEPOSITS FROM THE VALVES OR INTERIOR OF THE LEFT SIDE OF THE HEART, AND THEIR CIRCULATION WITH THE SYSTEMIC BLOOD

In endeavouring to elucidate this part of the subject, I beg to draw attention, in the first place, to instances in which it seems probable that masses of considerable magnitude have been detached from the left side of the heart, and subsequently arrested in an arterial channel of notable size; secondly, to some of the effects which seem to ensue when smaller arterial vessels or capillaries are similarly blocked up; and, thirdly, to circumstances which make it probable that, not unfrequently, the introduction of particles of fibrine into the circulating blood gives rise to constitutional symptoms indicative of a poisoned state of this fluid.

1. The first three cases which I shall offer are in many respects identical; for in each, death seemed to ensue from softening of the brain, consequent on obliteration of one of the main cerebral arteries by a mass of fibrinous material, apparently derived directly from warty growths on the left valves of the heart.

Case I.—Margaret Shaw, aet. 34, a pale, weakly-looking woman; admitted into St. Bartholomew's Hospital, under Dr. Roupell, about the middle of July, 1850, on account of pains in her lower limbs, and general debility. A loud systolic murmur was heard all over the cardiac region. No material change ensued in her condition until August 7th, when, while sitting up in bed eating her dinner, she suddenly fell back as if fainting, vomited a little, and when attended to was found speechless, though not unconscious, and partially hemiplegic on the left side. The hemiplegia increased, involving the left side of the face as well as the limbs, and gradually became complete in regard to motion, while sensation seemed to remain unimpaired. She continued speechless and hemiplegic, but without loss of consciousness, for five days, when she quietly died.

On examining the body, six hours after death, the skull and dura mater were found natural; but the small vessels of the pia mater were much congested, the congestion amounting, in some places, almost to ecchymoses. The right corpus striatum was softened to an extreme degree, being reduced to a complete pulp of a dirty greyish-white tint, and without any remains of its characteristic striated structure. The corresponding optic thalamus was healthy; but a condition of pale softening, similar to that affecting the corpus striatum, existed also to a considerable extent in the posterior lobe of the right cerebral hemisphere. The rest of the cerebral substance of this hemisphere was softer than natural, and appeared to contain less blood than ordinary. All other parts of the brain were healthy. The right middle cerebral artery just at its commencement was plugged up by a small nodule of firm, whitish, fibrinous-looking substance, which, although not adherent to the walls of the vessel, must have rendered its canal almost, if not quite, impervious. With the exception of a speck or two of yellow deposit in their coats, the rest of the vessels at the base of the brain were healthy and filled with dark blood.

The heart was enlarged; on its exterior were several broad white patches of old false membrane. The right cavities and left auricle contained recent separated coagula; the fibrine firm and whitish. The right valves were healthy; so also were the aortic, with the exception of slight increase of thickness. The mitral valve was much diseased, the auricular surface of its large cusp being beset with large warty excrescences of adherent blood-stained fibrine. There were a few scattered deposits in the coats of the aorta. The right common iliac artery, about an inch above the origin of its internal branch, was blocked up by a firm, pale, laminated coagulum, which extended into the internal iliac, and for about a quarter of an inch down the external iliac, where it terminated rather abruptly. The lower portion of the coagulum was colourless, and softer and more crumbling than the upper, which was also more blood-stained and laminated. There was no adhesion of the coagulum to the walls of the vessels. No similar clot existed in the iliac vessels on the opposite side. The pleurae were adherent in places; the lungs oedematous, and in places solidified by compact greyish-white masses, such as might result from uncured pneumonia. The pulmonary vessels were free from old coagula.

The liver and intestinal canal were healthy. The spleen was enlarged, pale, and soft. One large portion, about a fourth of the organ, was converted into a mass of firm, yellowish-white, cheesy substance. The kidneys were pale, rough, and granular. Within the cortex of the right were several large masses of yellow deposit, surrounded by patches of redness. The portions of medullary structure passing to these deposits were compact, dryish, and yellow.

In the case just narrated death evidently resulted from softening of a large portion of the right side of the brain; and the cause of this softening appeared to be an imperfect supply of blood, consequent on the middle cerebral artery of the same side being obstructed by a plug of fibrine within its canal. I am not aware that there has yet been recorded a case in which fatal softening of the brain resulted from a cause like this; therefore in itself this case is one of value. That the existence of the fibrinous coagulum within the cerebral artery was the real cause of the changes in the brain, can, I think, scarcely admit of question. The sufficiency of such an obstruction to produce the effects ascribed to it is fully established by the many instances in which disturbance, or complete arrest of function in a part, with subsequent atrophy or disorganisation of its tissue, results from any circumstance which materially impedes or entirely cuts off its supply of blood.

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PART II

ON THE EFFECTS WHICH MAY RESULT FROM THE DETACHMENT OF FIBRINOUS DEPOSITS FROM THE RIGHT VALVES OF THE HEART

If, from what has been stated, it be assumed as probable that deposits of fibrin occurring on the valves of the left side of the heart, may, by being detached, be productive of serious affections of remote organs, it may be inferred also that similar deposits occurring on the right valves may induce corresponding secondary affections of the lungs. And there seems to be sufficient evidence for believing that such is really the case. For it may, I think, be clearly shown, that most of the fibrinous or other similar secondary deposits in the lungs, also many of the old coagula found in the pulmonary artery or its branches, and possibly some forms of pulmonary apoplexy, are closely connected with it, if not actually dependent upon, fibrinous deposits on the valves, or interior of the right side of the heart, or materials transmitted through the heart by venous blood. It is of course conceivable that when the deposits on the right valves consist of large warty masses, as they occasionally do, portions of considerable size may be detached, and transmitted along the pulmonary artery, and so plug up one of the large branches of this vessel, just as similar masses detched from the left valves may be arrested in one of the main systemic arteries; but I have not yet met with a decided instance of such an occurrence. Probably the more usual manner in which the separation of fibrinous masses from the right valves leads to the formation of coagula in the pulmonary artery, is by the transmission of small particles to the minuter divisions of the artery, or to the capillary plexus, arrested at which they induce stagnation of the blood in those branches

of the artery distributed to the seats of obstruction. Such a result is almost necessarily consequent on the peculiar mode of distribution of the branches of the pulmonary artery, which pass to their destination without anastomosis. In a paper on the formation of coagula in the pulmonary artery, published in the Transactions of this Society, Mr. Paget has clearly shown the influence which certain obstructions in the pulmonary capillaries, such as oedema, chronic pneumonia, and pulmonary apoplexy, sometimes exercise in inducing coagulation of blood in the arteries supplying the obstructed parts. And I have likewise noticed a similar influence apparently resulting from other circumstances, such as extensive old tubercular disease, and extreme compression of the lung by false membrane on the pleura, which have obliterated large portions of the pulmonary tissue. Mr. Paget also narrates instances in which particles of cancerous matter brought from remote organs to the right side of the heart, and thence transmitted to the lungs, became arrested in the pulmonary capillaries, and so induced stagnation and subsequent changes of the blood, in branches of the pulmonary artery. Cases like these, of which I have seen several examples, seem to leave no doubt that a like coagulation of blood in the pulmonary arteries may result from obstruction caused by the arrest of particles of fibrine detached from the right valves of the heart, and transmitted to the pulmonary capillaries. An instance of this is furnished by one of the cases already narrated (Case III), in which, together with large, nodular, and warty masses attached to the tricuspid valve, nearly every branch of both divisions of the pulmonary artery were blocked up by old fibrinous coagula.

Another equally striking illustration is afforded by a specimen in the museum of St. Bartholomew's Hospital, in which, with extreme disease of the pulmonary valves, accompanied with the deposition of thick irregular layers of soft fibrine on each of them, there were old coagula filling many of the branches of the pulmonary artery. In this case there were also several large, solid, fibrinous masses in the substance of the lung; and it seems reasonable to believe that these, as well as the coagula in the pulmonary artery, had their origin in the deposits of fibrine on the pulmonary valves, portions of which were probably detached, arrested in the capillary plexus of the lungs, and so caused the fibrinous masses in the pulmonary tissue, and the consequent coagulation of blood in the arterial branches distributed to these parts.

The fibrinous masses in the lungs which the specimen just mentioned presents, appear not unlike portions of old pulmonary apoplexy, from which most of the colouring matter of the extravasated blood has been removed; and it is not improbable that many similar masses in other cases may have originated in a like cause, and not in haemorrhage into the pulmonary tissue. Such masses, indeed, represent one of the appearances described as capillary phlebitis of the lungs, or, in other words, one

stage in the transformation undergone by blood stagnant and coagulated in the pulmonary capillaries. This blood passes through the same changes in the lungs that it undergoes when similarly situated in other organs; and the various examples of these changes are not infrequently met with in the lungs. Thus, in Case III were found various gradations, from firm compact coagula, through soft, brownish, disorganised blood, to collections of yellowish, puriform material, which in places formed ordinary abscesses. Masses of such large size and with such obvious characters as these, are of course readily recognised. Yet not infrequently, deposits of a similar nature exist in the lungs, though of such extreme minuteness as to elude detection, unless specially sought for. These consist of small, slightly elevated, red dots, with a pale-yellow or buff-coloured centre, scattered, sometimes thickly, over the surface and within the interior of the lung. They are exactly identical with that spotted form of capillary phlebitis already mentioned as often occurring in systemic organs and in various tissues, either combined with other forms or alone. When met with in the lungs I have hitherto invariably found it either as the result of some morbid material in the venous blood, or in direct connection with affection of the right valves of the heart; such affection, namely, as is attended with the deposition of fibrinous granules on the surface of the valves. To quote but one instance out of several of the kind, I would mention the case of a girl under the care of Dr. Hue, early in the year 1851. This patient died suddenly, after suffering for some months with symptoms of extreme disease of the heart. Besides general enlargement of the heart, and narrowing of the mitral orifice, the free border of the tricuspid valve was studded with small, pale, fibrinous granules, a few of which existed also on the pulmonary valves. At first sight the lungs appeared healthy, but, on closer inspection, they were found freckled throughout with small, dark-red spots, like minute ecchymoses, in the interior of several of which was a distinct buff-coloured speck. The view which may not unreasonably be taken of these spots is, that they consisted of congested capillaries, in which minute fragments of fibrine, transmitted from the right valves of the heart had been arrested, the appearances, indeed being just such as resulted from the injection of softened meat into the blood in one of M. Gaspard's experiments.

Under whatever form these various deposits are met with in the lungs, I believe that careful examination will show them to be almost invariably associated either with the presence of fibrinous growths on the right valves of the heart, or with some other condition leading to the existence of particles of fibrine or other foreign matter in the blood transmitted to the lungs. Of these other conditions the most important seem to be the disintegration of old masses of fibrine situated within the right cavities of the heart, and a like disintegration of old coagula in some part of the

venous system, and its subsequent mixture with the venous blood. It appears to be quite usual for the old colourless or pale-reddish clots found in the right cavities of the heart, especially in the appendix of the auricle, to soften in the centre, and be converted into a dirty reddish-brown or fawn-coloured material. Sometimes the softening extends through the whole substance of the mass, with the exception of a thin layer at the circumference, which forms a kind of cyst or bag within which the softened material is contained. Sometimes too this cyst bursts and discharges its contents, leaving nothing but the outer shell attached to the interior of the heart. The softened material thus left loose and mingled with the blood will doubtless contaminate it almost as effectually as the direct introduction of a similar material by injection into a vein would do. And it is easy to imagine that the solid particles of fibrine may be arrested at the capillaries of the lungs, and produce the various forms, especially perhaps the spotted variety, of deposit to which allusion has been made. Old coagula in the veins too, under whatever circumstances they may have originated, appear almost equally liable to undergo softening, and to break up and mingle their disintegrated particles with the venous current along which they may pass to the lungs, and produce effects similar to those consequent on the transmission of like material from the cavities of the heart.

Such are some of the principal effects which the transference of fragments of fibrine from the right side of the heart appear capable of producing in the lungs. Much more might be said on the subject, but the length to which this communication has already extended precludes any further remarks at the present time. I would only add the suggestion that possibly the peculiar form of the pneumonia sometimes observed in rheumatic fever may, in some way, have its explanation in the transmission of fibrinous particles from the right valves of the heart to the lungs. The almost invariable existence of disease of the pulmonary or tricuspid valves in the fatal cases of rheumatic pneumonia I have examined after death strongly favours the opinion that there is some close relation between this peculiar inflammation of the lungs and the fibrinous deposits on the right valves of the heart.

In conclusion, let me briefly recapitulate the principal points I have endeavoured to establish to the satisfaction of the Society. They are, 1st, the general fact that fibrinous concretions on the valves or the interior of the heart admit of being readily detached during life, and mingled with the circulating blood; 2dly, that if detached and transmitted in large masses, they may suddenly block up a large artery, and so cut off the supply of blood to an important part; if in smaller masses, they may be arrested in vessels of much less size, and give rise to various morbid appearances in internal organs; while, under other circumstances, the particles mingled with the blood may be extremely minute, possibly the

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FATTY DEGENERATION OF THE HEART*

By

WILLIAM STOKES[†]

GENERAL DIAGNOSIS OF THE DISEASE

IF IT be inquired how far we have gone, since the time of Laënnec, in establishing the diagnosis of this affection, it will appear that as yet but little has been done. Laënnec declared that he knew of no means by which the diagnosis of fatty degeneration of the heart could be made; and Dr. Ormerod, writing in 1849, observed, that "the most extreme cases detailed may show that the diagnosis on general or physical grounds is almost impossible." "We cannot," he says in another place, "predict with certainty in any case that we shall find this lesion after death; but it is difficult for any pathological observer not to be led to suspect the existence of a disease in the repetition of the same circumstances under which he has seen it occur previously."

The diagnosis of this condition is not only possible but often free from difficulty, at least where the disease is confirmed. On the other hand, minor degrees of the affection are to be determined less by direct signs than by some general characters.

The diagnosis turns upon three points:—

1. The existence of physical signs and symptoms of diminished force of the heart.

2. The occurrence of certain symptoms, principally referrible to the brain, which indicate either anaemia on the arterial, or congestion on the venous side, of the cerebral circulation.

3. Symptoms referrible to the respiratory function, which appear to arise from deficient power in the right ventricle.

It is still to be determined how far we can distinguish during life the cases of weakened, and dilated hearts, such as have been already described, from those of fatty degeneration. Microscopical anatomy shows that in many of the former class there is more or less of the adipose deposit. And it is plain that to the practical physician there is a relation

momentary unsteadiness in walking, and in others a tendency to faint, which may be dissipated by any ordinary stimulus; while in the more decided cases the patient becomes suddenly comatose, a condition which may be preceded by loss of memory and a lethargic state. I have at present under my care a patient whose earlier attacks were syncopal; they are now apoplectic, and come on during sleep, each one being preceded by a slight convulsion. On recovery, and after all the comatose symptoms have passed away, he remains for half-an-hour unable to recognize his most intimate friends and relations, even his wife he has mistaken for his mother. This patient is 63 years of age. This latter symptom has been observed in a case of weak heart which lately occurred in Dublin; the patient frequently failing to recognize friends who had been his intimates for half a century. The duration of the attack is generally short, paralysis is rare, and when it occurs does not seem referrible to any anatomical lesion of the brain. The question as to whether these singular attacks are dependent upon deficient arterial supply, or rather upon venous congestion, is a difficult one, but it does not involve any important point of practice. It is true, that whatever arrests the action of the heart will retard the flow of blood in the veins of the head, but it could not cause a state of hyperaemia. The opinion that the apoplectic seizures are owing to deficient arterial supply seems the most tenable. The suddenness of the attack, and, in many instances, the rapidity of the recovery, are in favour of this view. I have noticed one case in which, on the occurrence of the premonitory symptoms, the patient, by hanging his head so that it rested on the floor, used to save himself from an attack. A case lately occurred to me of an aneurism of the aorta, in which three successive ruptures of the sac took place, with intervals of several days. Each rush of blood was attended with the best-marked syncopal coma and convulsions. Finally, dissection does not show any extra-ordinary congestion of the brain; and we learn from auscultation that the action of the heart is feeble.

This view of the cause of the attacks appears to be still further corroborated by the occurrence of symptoms of a similar nature in the case of a dilated mitral opening by Dr. Fleming, which has been already given. Here the ventricle was hypertrophied to a great degree, but the patient suffered from regurgitation into the left auricle.

We can, therefore, only adopt in part the plan of treatment suggested by the late Mr. Carmichael, which was to relieve the vessels of the head by venesection, while at the same time stimulants should be used to excite the action of the left ventricle.

Symptoms referrible to the respiratory function.—There is no evidence that the existence of this disease, even in an aggravated form, is an exciting cause of any organic affection of the lung. On the other hand, the

researches of Ormerod, Quain, and others, have demonstrated the frequent combination of fatty heart with pulmonary disease; but in such cases we may hold that the conditions of the lung and heart have little, if any mutual relation; they are rather to be considered as the secondary accidents of a general morbid state.

But there is a symptom which appears to belong to a weakened state of the heart, and which, therefore, may be looked for in many cases of the fatty degeneration. I have never seen it except in examples of that disease. The symptom in question was observed by Dr. Cheyne, although he did not connect it with the special lesion of the heart. It consists in the occurrence of a series of inspirations, increasing to a maximum, and then declining in force and length, until a state of apparent apnoea is established. In this condition the patient may remain for such a length of time as to make his attendants believe that he is dead, when a low inspiration, followed by one more decided, marks the commencement of a new ascending and then descending series of inspirations. This symptom, as occurring in its highest degree, I have only seen during a few weeks previous to the death of the patient. I do not know any more remarkable or characteristic phenomena, than those presented in this condition, whether we view the long-continued cessation of breathing, yet without any suffering on the part of the patient, or the maximum point of the series of inspirations, when the head is thrown back, the shoulders raised, and every muscle of inspiration thrown into the most violent action; yet all this without râle or any sign of mechanical obstruction. The vesicular murmur becomes gradually louder, and at the height of the paroxysm is intensely puerile.

The decline in the length and force of the respirations is as regular and remarkable as their progressive increase. The inspirations become each one less deep than the preceding, until they are all but imperceptible, and then the state of apparent apnoea occurs. This is at last broken by the faintest possible inspiration; the next effort is a little stronger, until, so to speak, the paroxysm of breathing is at its height, again to subside by a descending scale.

In other cases we see the symptom of sighing to occur in a different manner: at irregular intervals the patient draws a single deep sigh, especially when he suffers from fatigue, want of food, or of his ordinary stimulants. This is the commonest form of the affection.* In one case it was always most evident when the patient was lying down.

*The sighing respiration may be observed in persons who are labouring under certain forms of gastric or hepatic derangement, and is occasionally a symptom of undeveloped gout. It disappears under appropriate treatment, and probably indicates a temporary weakness of the heart. I lately saw a case of long-continued sighing, in which it had apparently arised from depression and anxiety of mind, but had, as it were, become a habit. The patient was a lady of very nervous disposition. A feeble murmur attended the first sound of the heart. In this case there was probably no organic lesion, for the symptom had long existed, and there were no signs of progressive disease.

The phenomena of circulation are next to be considered. We are in want of a sufficient number of observations to enable us to declare whether in the earlier periods there is any marked character of pulse as to strength, frequency or regularity. Many of the recorded cases of the minor stages of the disease are deficient in accurate observations of the pulse; but it may be held that no special character of pulse has been established. In some the pulse has been weak, rapid, and irregular; in others it does not seem to have differed materially from that of health.* But in confirmed cases we may meet with three important characters of pulse :—

1. The pulse somewhat accelerated, but occasionally intermitting; its strength may be but little altered.

2. The extremely weak, rapid, irregular, and tingling pulse (pulsus formicans).

3. The permanently slow pulse, the rate of which varies from 50 to 30 in the minute, or even less.

It is probable, that in the third class of cases, or those with a permanently slow, though distinct and regular pulse, the disease has either advanced to a great degree, or has at all events affected the different portions of the heart equally; and that we may attribute the weak and irregular pulse to conditions of the heart in which only certain portions of the organ have degenerated, or where there is a great difference between the right and left sides of the organ. It is further probable that the heart may be in two very different conditions previous to the commencement of the fatty change; and that in the case with irregular pulse, a merely weakened and perhaps dilated condition has preceded the deposit of fat globules in the muscular fibre; while in the third class the change

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Sufficient attention has not as yet been directed to this character of respiration. It is, when confirmed, almost pathognomonic of a weak and, in all probability, a fatty heart; but whether it is to be taken as indicative of the predominance of the fatty change on the right side of the heart is sti'l an open question. Laënnec has described a form of asthma with puerile respiration, and he attributes the disease and the signs to some special modification of the nervous influence. He observes, that he has never met with it except in persons affected with mucous catarrh, and holds that dyspnoea, arising from the mere increase of the natural want of the system for respiration, can never amount to asthma without the catarrhal complication. But he further speaks of adults and old persons who have puerile respiration without catarrh, and who, though they are not, properly speaking, asthmatic, are short-breathed, and liable to dyspnoea on the slightest exercise.

It is possible that in some of these cases at least, the heart may be in an incipient stage of fatty degeneration. I have observed the symptom in a gentleman of about 70 years of age, who has many symptoms of a weak heart. The action of that organ is regular, but the impulse is extremely feeble, and the pulse compressible. The sounds, especially the first, are very indistinct: there are no bronchial râles, but well-marked puerility of respiration exists over every portion of the thorax. He principally complains of dyspnoea on exercise, or any mental agitation; and the symptoms have only become prominent within the last eighteen months. So far as the permanent condition of the respiration is concerned, this case answers perfectly to Laënnec's description of dyspnoea with puerile respiration. See Dr. Forbes's translation of the work of Laënnec—Article, Asthma with Puerile Respiration.

*This circumstance is worthy of consideration in connection with that which I have recorded as occurring in cases of the softening of the heart in typhus, in many of which the pulse is quite a fallacious guide in determining the strength of the left ventricle.

has occurred without previous alteration in the structure or mode of action of the heart. Some of the cases observed in persons who have been long bedridden, and who have died from rupture of the left ventricle, are of this description. Additional observations, however, are necessary to elucidate this subject.

If we inquire whether irregularity of pulse is indicative of valvular disease in this affection, we must consider that the symptom may be met with in cases of weak, dilated hearts, without valvular disease, and therefore, that we might expect it in fatty degeneration. On the other hand, the occurrence of cases with a perfectly regular though slow pulse is a remarkable fact. In well-marked cases, where irregularity, rapidity, and smallness of pulse exist, we ought not, even though there be no valvular murmur, to declare too strongly against the existence of valvular obstruction; bearing in mind, first, that the very weakness of the heart may prevent the appearance of murmur; and next, that valvular disease is not infrequent combination with fatty heart. In most of the cases which I have seen, this valvular affection was at the aortic orifice, and the pulse was slow and regular.
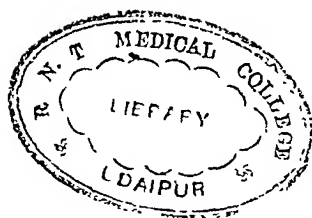
1861

PAUL LOUIS DUROZIEZ

DESCRIPTION OF THE DOUBLE INTERMITTENT
MURMUR OVER THE FEMORAL ARTERIES
IN AORTIC INSUFFICIENCY, LATER TO
BE KNOWN AS DUROZIEZ'S SIGN

PAUL LOUIS DUROZIEZ

(1826-1897)

"The Physician sees patients and not diseases."

—Duroziez, in Preface to
Traité Clinique des Maladies du Coeur, 1891.

$\mathcal{P}$AUL LOUIS DUROZIEZ was born in Paris on January 8, 1826. He was a student at L'Institution Favart, a Parisian grammar school. Later he became a student at the Lycée Charlemagne, where he excelled in Latin, Greek, and English, and in 1844 was graduated with the degree, Bachelor of Letters and Sciences.

Duroziez then began the study of medicine at the Faculté de Médecine of Paris and in the Paris hospitals. He studied under Alfred A. L. M. Velpeau (1795-1867) in 1845 and 1846. Later, in 1848, he became an extern to Dr. Blache at the Hôpital des Enfants Malades. In 1849 and 1850 he was an extern under Professor Jean-Baptiste Bouillaud (1796-1881) at La Charité in Paris. In 1850 he was awarded the Corvisart Prize for a clinical study of the subject, "Therapeutic Properties and Physiologic Action of Digitalis," the subject having been designated by the Faculty of Medicine. On July 21, 1853, he received the degree of Doctor of Medicine; his thesis was entitled: "Clinique de la Charité, Service de M. le Professeur Bouillaud, Sémestre d'Hiver 1850-1851."

In 1856 he was named chief of a clinic at La Charité in Bouillaud's service, where he remained until 1858.

His marriage to Mademoiselle Rohan, who was of Bourbon lineage and whose grandparents had been guillotined during the French Revolution, took place on June 25, 1861. Four children resulted from this marriage. The eldest, a boy, died at the age of eight from scurvy which developed during the siege of Paris in 1870. The other three children were daughters, each of whom received an excellent education. The year of his marriage (1861) Duroziez published his important paper on "The Double Intermittent Murmur over the Femoral Arteries as a Sign of Aortic Insufficiency." This murmur later became known as "Duroziez's sign." We are including an abstract, in translation, of this paper in our Cardiac Classics. Duroziez also elucidated in 1861 the pure type of mitral stenosis, setting forth its clinical character. This later became known as "Duroziez's disease."[1]

In 1867, Duroziez was appointed physician of the Bureau of Welfare of the First Ward of Paris. During the Franco-Prussian War, in 1870, he served as ambulance physician and surgeon major of the Fourteenth Infantry Battalion. For his outstanding work with patients during the smallpox epidemic he was awarded a silver medal from the Minister of the Interior.

In 1879, he was appointed a member of the Commission of Public Hygiene and Health of the First Ward of Paris, and in 1889 he was appointed medical inspector of schools of the same ward. In 1891, he was awarded the Itard Prize of the Academy of Medicine for his work, "Traité Clinique des Maladies du Coeur." In 1891, also, L'Institut de France awarded him the Montyon Prize for the same work.

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[1]Duroziez, P.: Du rétrécissement mitral pur, Arch. gén. de méd. 140: 32-54, 1877.

At the age of seventy, on January 4, 1895, Duroziez was named a chevalier of the Legion of Honor. The honor came when he had ceased to care much about it, and to the patient who brought him the news he exclaimed: "What a good thing for my wife!"

For thirty years Duroziez occupied an important place in the Société de Médecine, before which body he made many original contributions which were subsequently published in the "Communications" of that society. He was its president in 1882.

Early in 1897, Duroziez contracted pneumonia, to which he succumbed at noon on January 16, 1897.

Duroziez always professed a profound and genuine interest in the diseases of the heart. "As long as my own heart beats," he said to a contemporary, "I shall continue to auscultate the one of others." He considered that organ as a separate being endowed with a male half, the left ventricle, and a female half, the right ventricle. The former, he thought, was calm, regular and stable; the latter, he considered, was nervous, impressionable, and often disordered. In his original work on the duality of the heart[2] Duroziez referred to the four cardiac cavities, comparing them to four horses fastened to the same chariot. He pointed out that such an arrangement permitted an easy break in equilibrium with resulting badly combined movements.

Duroziez once remarked, "In the heart, bruits are too full of detail, too brief. The cavities do not always have the same relationship. One does not know where one is." In spite of this remark, Helfenbein wrote that Dr. Jullien said of Duroziez, "Bruits were to him sweet music; he listened to them in the chest, surprised them in the back, pursued them into the neck and even into the thigh."

Duroziez also introduced the onomatopoetic Fout-tata-Rou which he used to describe the various signs in the heart heard in mitral stenosis. Among his many observations, mention should be made of his description of the sequelae of pure mitral stenosis, such as embolism, aphasia, and right hemiplegia. He also noted the predominance of this disease among women.

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[2]Duroziez, P.: De la dualité du coeur, Bull. Soc. de méd. de Paris 27: 39-42, 1893.

THE DOUBLE INTERMITTENT MURMUR OVER THE FEMORAL ARTERIES AS A SIGN OF AORTIC INSUFFICIENCY*

By

DR. P. DUROZIEZ

Former chief of the Clinic of the Faculty at the Charité Hospital
(Service of Professor Bouillaud)

. . . .

The femoral arteries, which are subjected to auscultation less frequently than the carotids, offer very valuable information; they are especially unique in their behavior, they are readily compressed and in this respect offer the same advantages as the radial arteries: they are larger than the carotid arteries and have the advantage of being more distant from the heart. . . .

The femoral artery merits careful study.

On compression of the femoral artery, a shock or thrill is felt and auscultation reveals a sound, similar to the sound of *toc* or a sound of unique blowing character, a simple intermittent blowing murmur. The entire femoral artery is capable of giving rise to this blowing murmur. The character of the murmur varies with changes in the blood, the size of the artery, the condition of the vessel wall, and the contractile force of the heart. After compressing the artery for some time and gradually releasing the pressure in a subject with chlorosis, a continuous humming murmur will appear; at times a continuous, humming sound is audible, at other times a double murmur is audible.

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The so-called intermittent double murmur which occurs in certain cases, is a different murmur and our study will concern it. . . .

The intermittent double murmur over the femoral arteries was described in aortic insufficiency; but no one, I believe, has given it the significance that it deserves. Everyone has mentioned the murmur occurring in arterial diastole (*souffle de la diastole arterielle*) which quite frequently occurs without compression of the artery; but very few authors mentioned the murmur occurring during systole. Very frequently it does not appear of its own accord, but must be produced and sought for. The

first murmur results from the powerful contraction of the ventricle, but as the second murmur is produced by the systole of the arteries in the legs, a less powerful force, its production must be facilitated by compression of the artery.

In cases of uncomplicated aortic insufficiency, wherein the heart beats vigorously and the arteries pulsate and react forcefully, the double murmur is audible; when, contrarily, aortic insufficiency is complicated by a considerable degree of aortic or mitral stenosis, a not uncommon occurrence, the arteries are moderately distended with blood and thus the second murmur is difficult to hear. It must be carefully sought and even then it will not appear regularly; it will not be detected when weak pulsations are present. It appears or disappears in relationship to increased or decreased action of the heart. At times it can be heard over both femorals, at other times only over one; briefly, distention and recoil, adequate systole of the arteries, are required for its presence; a careful examination is indispensable.

The double murmur can be produced in two ways, by means of the stethoscope or by means of the hand. With the stethoscope pressure is exerted to completely compress the artery; at a certain moment the double murmur will appear; only when the second murmur can be readily produced is it possible to place the stethoscope on the artery without pressure and then gradually slight pressure can be exerted with the hand above and below the stethoscope. Pressure above will produce the first murmur, while pressure below will produce the second murmur; it is evident that the second murmur is produced by the arteries of the legs, which propel the blood backwards and in some manner empty the capillaries.

The double intermittent murmur is of interest not only from the standpoint of diagnosis. The reflux of blood explains some of the symptoms occurring in aortic insufficiency and explains the sudden death which is occasionally observed.

A great disturbance occurs in the circulation; the blood no longer circulates evenly, so to speak, but comes and goes into the arterial system and stagnates in the veins, which continuously try to empty themselves. In the presence of aortic insufficiency, the heart during its powerful diastole, aspirates the blood from the lungs through the pulmonary veins at the same time that it receives the blood from the capillaries; the right ventricle and the lungs are emptied of blood. The blood supply is poor; the patients are pale, die from anemia and syncope. They do not tolerate venesection well.

What a difference occurs with mitral stenosis! Here, on the contrary, the blood is stagnant, forced into the veins, into the right side of the heart and the lungs; the patients die from apoplexy and suffocation; venesection gives relief.

These are two conditions, in opposition to each other, and one may be considered as being beneficial to the other.

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Auscultation is an important issue in this connection. We have distinguished by our observations the auscultatory phenomena and particularly those concerning the femoral arteries.

1. In all heart cases wherein the double intermittent murmur was audible over the femoral arteries, aortic insufficiency was found at autopsy.

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Conclusions

1. The double intermittent murmur audible over the femoral arteries, described by many authors in aortic insufficiency, has to my knowledge never been given as a constant sign of this lesion.

2. Most commonly it is not present and it is necessary to produce it by compression.

3. In aortic insufficiency blood is first propelled from the left ventricle into the extremities, and, being repulsed by the peripheral arteries and drawn back by the left ventricle, flows from the extremities towards the heart.

4. The finger, compressing the artery about two centimeters above the stethoscope, produces the first murmur; two centimeters below, the second murmur.

5. The secondary murmurs which can be produced by lesions of the pericardium, by mitral stenosis, tricuspid stenosis, by pulmonary insufficiency, can be differentiated from the murmur of aortic insufficiency with the help of the double murmur over the femoral arteries, which exists only in the latter condition.

6. If aortic insufficiency is complicated by one or more of the lesions mentioned, and if the diagnosis is rendered difficult by these complications, the phenomena in the femoral arteries will help or even establish the diagnosis.

7. The femoral phenomena less clearly differentiate aortic valvular lesions and lesions of the aorta. The double murmur may appear in certain aneurysms without insufficiency being demonstrable after death.

8. The temporary insufficiency can be demonstrated by the evanescent intermittent double murmur.

9. A continuous murmur can originate in the arteries; this, however, is never audible in aortic insufficiency with its constant intermittent double murmur over the femoral arteries.

10. The double intermittent femoral murmur occurs in typhoid fever, chlorosis, lead intoxication, but only temporarily; it is soon replaced by continuous murmurs.

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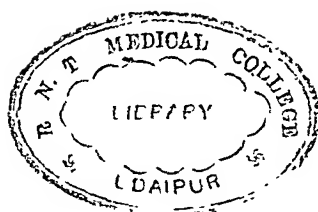
1862

AUSTIN FLINT

DESCRIPTION OF THE MURMUR LATER TO BE
KNOWN AS THE AUSTIN FLINT MURMUR

AUSTIN FLINT

(Courtesy Charles C Thomas.)

cine, as well as clinical medicine, until 1852, when he resigned to become professor of the theory and practice of medicine at the University of Louisville.

Flint served four years at Louisville after which (1856) he returned to Buffalo where he accepted the chair of pathology and clinical medicine in the school he had helped to found. Flint spent the winter seasons from 1858 to 1861 at New Orleans, Louisiana, where he filled the professorship of clinical medicine at the New Orleans School of Medicine and was attending physician to the Charity Hospital.

In 1859 Austin Flint accepted two faculty appointments in New York City. He became professor of the principles and practice of medicine and clinical medicine and visiting physician at Bellevue Hospital Medical College. He also accepted the professorship of pathology and practical medicine at the Long Island College Hospital. He resigned from the latter position in 1868.

Flint was a member of many medical and scientific societies in America and Europe. He was elected to the presidency of the New York Academy of Medicine for the term, 1873-1874. In 1883 he was elected president of the American Medical Association, an office which he had not sought and which he did not desire. He had been made chairman of the Section on Practical Medicine in 1850, and in fact, had helped to found the Association in 1848.

At the meeting of the International Medical Congress in London in 1881, Dr. Flint read a paper on ''The Analytical Study of Auscultation and Percussion with Reference to the Distinctive Characteristics of the Pulmonary Signs.'' This paper was received with such acclaim that Flint was asked to serve as chairman of a newly formed committee which was to report on a ''Uniform Nomenclature of Auscultatory Sounds in the Diagnosis of Diseases of the Chest.'' The committee's report was made at Copenhagen in 1884. Flint's suggestion, made with Dr. Samuel D. Gross, led to the decision that the International Medical Congress should convene in 1887 in the United States. Flint was to have succeeded Samuel D. Gross as president of this body, but Gross died in 1884, on the very day on which Flint, as president of the American Medical Association, asked that the Congress meet in America. Flint was elected president of the Congress, but died in 1886, before it met.

Austin Flint was a prolific writer on all phases of medicine. It is outside the scope of this brief sketch to list all his contributions to the literature, but mention may be made of his outstanding contributions to physical diagnosis and to his original observations on diseases of the heart and lungs.

Neither Laënnec nor his immediate followers paid any attention to changes in the pitch of percussion notes or respiratory sounds. Credit for this important addition to the art of physical diagnosis belongs to Austin Flint. His observations were embodied in an essay entitled, ''The Variations of Pitch in Percussion and Respiratory Sounds, and Their Application to Physical Diagnosis.'' This essay was awarded the annual prize of the American Medical Association for 1852. Flint also won the first prize of the American Medical Association in 1859 for his essay, ''The Clinical Study of the Heart Sounds in Health and Disease.'' This, too, was the year he published an important book on diseases of the heart.[1]

Flint's name is most frequently thought of in its association with the presystolic murmur which sometimes accompanies aortic regurgitation. This he was the first to describe. Flint heartily disapproved of associating any physical sign with the name of the original describer. He wrote:[2] ''So long as signs are determined from fancied analogies, and named from these or after the person who describes them, there cannot but be obscurity and confusion.''

[1]Flint, Austin: *A Practical Treatise on the Diagnosis, Pathology, and Treatment of Diseases of the Heart*, Philadelphia, 1859, Blanchard and Lea, 473 pp.

[2]Landis, H. R. M.: Austin Flint; his Contributions to the Art of Physical Diagnosis and the Study of Tuberculosis, Bull. Johns Hopkins Hosp. 23: 182-186, 1912.

The first time he observed the so-called Flint murmur was in 1859, in examining a patient in Charity Hospital in New Orleans who had well-marked signs of aortic insufficiency and stenosis and in whom a presystolic murmur was audible at the apex. At necropsy, however, the mitral valves were found to be normal. This classic description was published in 1862 in the "American Journal of the Medical Sciences," and it is our privilege to reprint it here.

Flint was by no means a specialist. His contributions to the study of tuberculosis are among the best in American medical literature. His first paper on this subject was published in 1849. In this early article Flint emphasized the importance of recognizing the disease in its incipient stages, for as he said, on this factor depended control of the disease.

A masterful treatise on tuberculosis was his "Phthisis," published in 1873.[3] The work is an analysis of 670 cases. Landis said that it deserved to be ranked with the great work of Pierre C. A. Louis on tuberculosis.

One of the distinguishing features of Austin Flint was the receptiveness of his mind to new ideas. Shortly after Koch's momentous discovery of the etiology of tuberculosis in 1882, Flint began having the sputum of patients at Bellevue Hospital examined. From his study of these patients he immediately saw the enormous im-importance of Koch's discovery. In a paper[4] read in his seventy-second year, he emphasized the fact that tuberculosis could be contracted by a normal person's exposure to the disease.

Flint's last article, which was published posthumously, was his "Medicine of the Future."[5] Therein he foresaw that physiologic and pathologic chemistry must be investigated to explain many of the phenomena of health and disease. He had already accepted as valid the role of bacteria in health and disease and he foretold great progress in the understanding of this branch of medical science.

Austin Flint's death was sudden and unexpected. He had attended a meeting of the faculty of the Bellevue Hospital Medical College on Friday evening, March 12, 1886. On returning home he proceeded to retire and without any warning a cerebral hemorrhage occurred. This was followed by unconsciousness, resulting in Flint's death fourteen hours later, on Saturday, March 13, 1886.

[3]Flint, Austin: *Phthisis; its Morbid Anatomy, Etiology, Symptomatic Events and Complications, Fatality and Prognosis, Treatment and Physical Diagnosis, in a Series of Clinical Studies,* Philadelphia, 1875, H. C. Lea, 446 pp.
[4]Flint, Austin: *On the Pathological and Practical Relations of the Doctrines of the Bacillus Tuberculosis,* 16 pp. Reprinted from Med. News, Philadelphia, 1884, vol. 440
[5]Flint, Austin: *Medicine of the Future.* An address prepared for the annual meeting of the British Medical Association in 1886. New York, 1886, D. Appleton & Co., 37 pp.

ON CARDIAC MURMURS*

By

AUSTIN FLINT, M.D.

*Professor of the Principles and Practice of Medicine in the Bellevue Hospital
Medical College, N. Y., and in the Long Island College Hospital*

THE clinical study of cardiac murmurs, within the last few years, has led to our present knowledge of the diagnosis of valvular lesions of the heart. By means of the organic murmurs it is positively ascertained that lesions exist in cases in which, without taking cognizance of the murmurs, the existence of lesions could only be guessed at. The absence of the organic murmurs, on the other hand, enables us generally to conclude with positiveness that valvular lesions do not exist. As a rule, to which there are but few exceptions, these lesions may be excluded, if there be no murmur. These are great results; but the practical auscultator of the present day need not be told that the clinical study of cardiac murmurs has led still further into the mysteries of diagnosis. Having ascertained the different murmurs which occur in connection with valvular lesions; having traced their connection, respectively, with different lesions; having shown their relations to the movements of the several portions of the heart, and to the cardiac sounds;[1] and, having explained satisfactorily the mechanism of their production, we are able to determine not only the existence or non-existence of valvular lesions, but their particular situation when they are present, and, to a certain extent, their character and consequences. The practiced auscultator, by listening to the murmurs alone, is able to tell whether lesions are situated at the mitral, or the aortic, or the pulmonic orifice, and he is able to say, in certain cases, that the valves which are to protect these orifices against a regurgitant current of blood, have been rendered by disease inadequate to their office. It is unnecessary to adduce proof of these statements; their correctness is sufficiently known to those who are conversant with physical exploration as applied to the diagnosis of affections of the heart. How strikingly do these facts exemplify the progress of practical medicine to those who, although still among the junior members of the profession, have practised before and since the recent developments in this department of our knowledge!

[1]The conventional distinction between the cardiac *sounds* and *murmurs* is to be borne in mind; the former term being limited to the normal heart-sounds with their abnormal modifications, and the latter to newly-developed or adventitious sounds, which are altogether the products of disease.

These remarks are introductory to the consideration of various practical points pertaining to the cardiac murmurs. And the first subject will relate to these murmurs in general—viz., *the limitations of their significance*. After having considered certain points embraced in this subject, I propose to take up various points relating to the different murmurs separately.

By the limitations of the significance of the murmurs, I mean the actual amount of knowledge respecting valvular lesions to be derived from this source. It is evident, from what has been stated already, that the knowledge which they convey is of very great importance, but important as this knowledge is, it has certain limits which are not always sufficiently understood; and, as a consequence, the practitioner is liable to fall into unfortunate errors of opinion as regards the gravity of the lesions which the murmurs represent.

Prior to the clinical study of the cardiac murmurs, the existence of organic affections of the heart was recognized when, in conjunction with disturbed action of the organ, symptomatic events had taken place which *belong to an advanced stage of only a certain proportion of cases*. Dyspnoea, palpitation, and dropsy, were the symptoms mainly relied upon for the diagnosis. The recognized cases were then comparatively rare, and, when recognized, a speedily fatal issue was expected. This fact, together with the frequency with which cardiac lesions were revealed by postmortem examinations in cases of sudden death, rendered the diagnosis of organic disease of the heart equivalent to a summons from the grave. The prognosis, as a matter of course, was as unfavourable as possible; the doom of the patient was either to die unexpectedly at any moment, or to endure protracted sufferings until released by death. The study of the murmurs together with the application of other signs, enabled the practitioner to recognize organic affections at a period in the disease when otherwise they would not have been discovered. The recognized cases became more frequent. Persons were found to have cardiac lesions who presented few or no symptoms pointing obviously to disease of the heart. The ideas which had prevailed relative to the gravity of organic affections, however, naturally enough, continued to prevail. An organic murmur, consequently, had a fearful significance. It was considered as proof of disease which was not less surely destructive because earlier ascertained. Let it be said of a patient that he had a cardiac murmur denoting a valvular lesion, and his doom was pronounced; sudden death, which might occur at any time, or an early development of the distressing symptoms characteristic of cardiac disease, were to be expected, whatever might be his present condition.

So far from concealing from patients the fearful significance of cardiac murmurs, it was considered important for them to understand fully their precarious condition, in order to receive their co-operation in the meas-

ures of management which were deemed essential. These measures embraced general and local blood-letting, depletion by cathartics, sedative remedies addressed to the circulation, mercurialization, low diet, together with as much inaction of mind and body as possible. The consequences of this management were calamitous in the extreme. In fact, these measures contributed, in no small degree, to the fulfilment of the gloomy predictions impressed upon the minds of the unfortunate patients who were found to present the auscultatory sign of valvular lesions. So long as these notions with regard to the treatment of cardiac affections prevailed, an early diagnosis, instead of being desirable, was a serious disadvantage, and truly fortunate were they who kept aloof from the stethoscope of the auscultator!

Erroneous views respecting the significance of cardiac murmurs, and also respecting the indications for treatment in cases of organic disease of the heart, are still, to a greater or less extent, prevalent. I propose now to confine myself to the former, i.e., the significance of the murmurs. It is obvious that with the acquirement of means of ascertaining the existence of lesions at an early period, when, without these means, the lesions could not have been discovered, clinical experience had to take a new point of departure as regards prognosis. And experience has shown that lesions giving rise to cardiac murmurs by no means invariably denote impending danger or serious evils, and that they are not unfrequently innocuous. Several clinical observers, within the last few years, have contributed facts going to show the correctness of this statement. Of these, Dr. Stokes[1] is especially prominent. Dr. Gairdner, of Edinburgh, has lately communicated a valuable paper on this subject.[2] I have been able to gather some facts having an important bearing on the subject under consideration. Of the cases which have come under my observation, exemplifying the "limitations of the significance of cardiac murmurs," I shall select a few of the most striking.

Thirteen years ago, I attended a child, aged 11 years, with a slight rheumatic attack. Directing attention to the heart, I found a very loud mitral, regurgitant murmur, heard over the left lateral surface of the chest and on the back. The heart was enlarged, the extent and degree of dulness in the praecordia being increased, and the apex beat without the nipple. The murmur was evidently not due to an endocarditis developed during the present attack of rheumatism; the lesion giving rise to it probably originated in an obscure thoracic affection which had occurred seven years before. I was at that time less acquainted with the significance of cardiac murmurs than now, and I deemed it my duty to inform the mother of the patient of the existence of an organic affection of the heart, which would be likely to destroy life within a period not

[1]Diseases of the Heart and Aorta.
[2]Edinburgh Monthly Journal of Med. Science.

very remote. The patient is still living. She is now 24 years of age, and, although presenting a delicate appearance, a casual observer would not suspect the existence of any disease. She is subject to palpitation, to coldness of the extremities, and experiences want of breath on active exercise, but she does not consider herself an invalid, and the apprehensions caused by my communication to the mother have long since disappeared.

It is fair to presume that my opinion in this case was considered as a mistake. It was, indeed, an error of judgment as regards the prognosis, but the diagnosis was correct; the loud bellows murmur is still there, and heard over the whole chest, even through the dress, and the heart is considerably enlarged. The patient, if not destroyed by some intercurrent affection, will ultimately die of cardiac disease, yet it is now twenty years since the probable commencement of the lesions giving rise to the cardiac murmur.

Nearly twenty years ago a person was examined by a medical friend with reference to an assurance on his life. My friend, finding a loud murmur, and an abnormally strong action of the heart, brought the person to me as an interesting case for examination. I failed to record the case, and am not therefore positive as regards the particular murmur present, but I think it was the mitral regurgitant. Since that examination, until recently, I have been in the habit of meeting this person often, although he has never been my patient. He has been, and still is engaged in active business. He is now about fifty years of age. He has survived his wife, and been again married within a few years.

I have selected these two cases as illustrating the duration of life and comfortable health for thirteen and twenty years after a loud organic murmur, together with enlargement of the heart, had been ascertained; in both cases life and comfortable health continuing at the present moment. I could cite, in addition, numerous cases of persons now living, and apparently well, who have had organic murmurs for several years. In making examinations of chests, supposed to be healthy, for purposes of study, I have repeatedly found a murmur, evidently organic, when no disease of the heart was suspected either before or after my examination. The following case is instructive, as showing the importance of taking into account the coexistence of functional disorder of the heart, dependent on anaemia, with organic disease.

In November, 1852, I visited, in consultation with Professor Rogers, of Louisville, a lady aged about 25. She had had repeated attacks of acute rheumatism. She had an infant several months old, which she was nursing, and she had become quite anaemic. She had begun to suffer from palpitation during the preceding summer, and her attention was attracted to a sound in the chest which she heard in the nighttime. This sound was also heard by a sister with whom she slept. She described, of her own accord, the sound to be like that produced by a pair of bellows.

She had never heard of cardiac bellows-murmurs, and at this time there had been no examination of the chest. Prof. Rogers had been called to the patient a short time before my visit, and detected at once the existence of organic disease.

She presented an aortic direct and a mitral regurgitant murmur, both loud; the heart was moderately enlarged, and its action violent. She was conscious of this violent action, and slight exercise or mental excitement occasioned much distress from palpitation. The urgent symptoms in the case were attributed to anaemia; weaning was at once enjoined, and chalybeate remedies, etc., advised. I met the patient a year afterwards without recognizing her. She was apparently in perfect health, and presented a blooming appearance. Her friends thought we must have been mistaken in our opinion as to the existence of organic disease of the heart. The murmurs and the signs of enlargement, however, were still there. She continued to enjoy good health until the summer of 1856, when she suffered from uterine hemorrhage, and again became anaemic. The action of the heart became irregular, and she complained much of vertigo. Tonics, stimulants, nutritious diet and fresh air failed to remove the anaemic state, and at length she was seized with apoplexy and hemiplegia. She recovered from the apoplexy, but the hemiplegia continued, and death took place between two and three weeks after the apoplectic seizure.

The significance of organic murmurs is limited to the points of information already stated in the introductory remarks, viz., the existence of lesions, their localization, and the fact of valvular insufficiency or regurgitation. Whether the lesions involve immediate danger to life, or, on the contrary, are compatible with many years of comfortable health, the murmurs do not inform us, nor do they teach us how far existing symptoms are referable to the lesions, and how far to functional disorder induced by other morbid conditions. Neither the intensity nor the quality of sound in the murmurs furnish any criteria by which the gravity of the lesions or their innocuousness can be determined. A loud murmur is even more likely to be produced in connection with comparatively unimportant lesions than with those of a grave character, because in the former, rather than in the latter case, is the action of the heart likely to be strong, and the intensity of the murmur, other things being equal, will depend on the force with which the currents of blood are moved. Whether the murmur be soft, or rough, or musical, depends not on the amount of damage which the lesions have occasioned, but on physical circumstances alike consistent with trivial and grave affections.

It may be imagined that these assertions, although true as regards murmurs produced by the direct currents of blood, do not hold good with respect to the regurgitant murmurs. The latter, it may be said, involving as they do insufficiency of the valves, will be loud in proportion

to the amount of blood which regurgitates, and, therefore, the intensity of the murmur should be a criterion of the amount of valvular insufficiency. But clinical observation disproves this surmise. A minute regurgitant stream is as likely to be intensely murmuring as a large current, perhaps even more so. Here, too, the loudness of the sound will depend, in a great measure, on the power of the heart's action. To this point I shall recur when I come to consider the different murmurs separately.

The practical injunction to be enforced in connection with the limitations of the significance of the cardiac murmurs is, that we are not to judge of the magnitude of valvular lesions, of the amount of danger on the one hand, or of the absence of danger on the other hand, by the characters belonging to the murmurs. The physician who undertakes to interrogate the heart by auscultation is not to decide that the condition of his patient is alarming, simply because he finds a murmur which he satisfies himself is dependent on an organic lesion of some kind. The lesion may be at that time, and perhaps ever afterwards, innocuous; the evils arising from cardiac affections may be remote, and so far from plunging the patient into despair by the announcement of the fact that he has an incurable disease of the heart, there may be just grounds for holding out expectations of life and comfortable health for an indefinite period. Neither does it necessarily alter the case when more than one murmur is discovered; the existence of several murmurs by no means excludes the possibility of similar encouragement. We are to look to other sources of information than the murmurs in forming an opinion respecting the gravity of the affection. What are the sources of information on which our opinion is based? It does not fall within the scope of this essay to consider at length the points involved in the answer to this inquiry. I shall answer it in a few words.

The heart-sounds furnish means of determining whether the lesions are of a nature to affect materially the function of the valves. I must here pass by this useful and beautiful application of auscultation with a simple allusion to it, referring the reader elsewhere for a full exposition of the subject.[1] I shall, however, return to the subject presently in considering the murmurs individually. Means requiring less proficiency in physical exploration relate to enlargement of the heart. It is not a difficult problem to determine whether the heart be or be not enlarged, and it is easy to determine the degree of enlargement. Now, in general, if valvular lesions have not led to enlargement of the heart, they are not immediately dangerous, and the danger is more or less remote. Here is a criterion of great importance in estimating the gravity, on the one hand, or the present innocuousness on the other hand, of lesions giving rise to murmurs. So long as the heart be not enlarged, the lesions cannot have occasioned to much extent those disturbances which arise from contrac-

[1]Clinical Study of the Heart-sounds in Health and Disease. Prize Essay, Trans. Amer. Med. Association, 1859.

tion or patency of the orifices. The murmurs, in themselves, give no information respecting the amount of obstruction from contracted orifices, or of regurgitation from valvular insufficiency. Let this fact be constantly borne in mind. But obstruction and regurgitation, singly or combined, inevitably lead to enlargement of the heart; hence the latter becomes evidence of the former. The degree of enlargement is, in general, a guide to the extent and duration of the disturbances occasioned by contracted and patescent orifices. As a rule, if the heart be slightly or moderately enlarged, the immediate danger from the lesions which may give rise to one or more loud murmurs is not great.

The truth is, the evils and danger arising from valvular lesions, for the most part, are not dependent directly on these lesions, but on the enlargement of the heart resulting from the lesions. We may go a step further than this and say that, ordinarily, serious consequences of valvular lesions do not follow until the heart becomes weakened either by dilatation or by degenerative changes of tissue. So long as the enlargement be due mainly to hypertrophy of the muscular walls, the patient is comparatively safe. Hypertrophy is a compensatory provision, the augmented power of the heart's action enabling the organ to carry on the circulation in spite of the disturbance due to obstruction and regurgitation. Happily, in most cases, hypertrophy is the first effect of valvular lesions, and, for a time, it keeps pace with the progress of the latter. Dilatation which weakens the heart's action, is an effect consecutive to hypertrophy, and, as a rule, it is not until the dilatation predominates that distressing and dangerous evils are manifested.

The practical bearing of these views respecting hypertrophy and dilatation, on the management of organic affections of the heart, is obvious. They are inconsistent with the employment of measures to prevent or diminish hypertrophy; on the contrary, they point to the importance of an opposite end of management, viz., to encourage hypertrophy in preference to dilatation, and to maintain the vigour of the heart's action. It does not fall within the scope of this essay to consider therapeutical applications, and I must content myself with this passing notice of an immensely important reform in the management of organic affections of the heart.

Returning to the means of determining the gravity of valvular lesions, I repeat, they become serious, in other words, the distressing and dangerous symptomatic events are to be expected, in proportion as hypertrophy merges into dilatation, or as weakness of the organ may be induced by structural degeneration or other causes. In connection, then, with murmurs, we are to determine the condition of the heart as respects the points just mentioned, in order to estimate properly the gravity of the lesions which the murmurs represent. In leaving this subject, viz.,

the limited significance of the cardiac murmurs, I will give a case which is a type of a class of cases not infrequently coming under observation.

In the spring of 1860, I was consulted by a medical gentleman from a distant State, who furnished me with the following written statement of his case:—

"About a year ago I went to the city of ——— to place myself under the care of Dr. ———, for a trifling surgical difficulty with which I had been annoyed for a long time. At long intervals previous to that time I had had severe pains in the left breast about the cardiac region, but at no time from any constant pain. I thought the pain was of a neuralgic character. While at ——— I thought I would have my lungs examined, as some members of my family had been consumptive. I went to Dr. ——— and to Dr. ———, both of whom pronounced my lungs sound, but said that my heart was affected. I came home much depressed by their opinion, and suffered so much from mental anxiety that in the course of a month or two I determined to go back and consult another medical gentleman, Dr. ———. He told me there was some roughness about the sounds of the heart but no serious organic disease. I was much relieved by this opinion, and clung to the belief that the pains were of a neuralgic character.[1] Previous to my going to ——— I had all my life taken a good deal of out-door exercise, such as riding, hunting, fishing, etc., for the purpose of warding off any tendency to consumption. I have always had a frail figure and been inclined to despondency. I have suffered a great deal of anxiety, owing to family affairs and business matters. After my return from consulting Dr. ——— I thought it best to give up active exercise for fear of increasing any cardiac affection that might exist. I do not think that I have had any severe pain in my chest frequently, at any time, but only at intervals and apparently occasioned by anxiety about patients, etc.

"In December last I went into the country, 13 miles to see a patient. The weather was very cold, rainy, and windy; I returned in the night. I was suffering from toothache and smoked a cigar in order to relieve the pain. I went over to my office to write a prescription for a sick child, and on my way back I was attacked by palpitation of the heart for the first time in my life. I came into the house and lay down, when I was seized with severe rigors without chills. I had also pain in the back, and afterwards fever. Since then I have been subject, at intervals, to a jarring or knocking sensation about the heart, but no palpitation of long continuance. I cannot sleep as well on my left side as formerly, as it causes an uneasy sensation with something like palpitation and some pain. I do not take much exercise, and find that I get out of breath easily. I am very sensitive to cold. The attacks of increased action of the heart are always accompanied by rigors and irritability of the bladder. On the 19th of March, I was taken with a feeling of fatigue and indigestion, followed by severe rigors together with great heat of the head and body. The circulation was rapid and accompanied by palpitation. The attack lasted nearly an hour, and I feel the effect of it today, March 22d. I notice, when reading a newspaper or small book, that the action of the heart causes it to vibrate. During my first attack in Dec., I had

[1]Doubtless they were so.

an intermittent pulse. I did not recover from that attack so as to go out for a week, and have not since been as well as before.

"Fearing that my situation was critical I have been careful of myself. I have feared to increase the affection and that I might die suddenly. But I have had fear that in taking care of the cardiac affection I shall increase a tendency to consumption. Any mental anxiety increases the action of the heart. I do not smoke nor chew tobacco, nor drink any alcoholic liquors. I have suffered much from toothache; in other respects have had generally very good health. I have never had rheumatism. I am a married man with five children. I think my cardiac affection has been getting worse since December last, and I suffer in mind dreadfully on that account, as I have a great deal to live for."

On examination of the chest, in this case, I found the apex-beat in the 5th intercostal space half an inch within a vertical line passing through the nipple. The area of superficial cardiac dulness carefully delineated on the chest, was found to be of normal extent. The left border of the heart fell within the nipple. The respiratory murmur, on a deep inspiration, was heard over the whole praecordia. The apex-beat was not abnormally strong; no other impulse was discovered, and no heaving of the praecordia.

At the first examination, the heart being but little excited, I discovered a slight murmur just to the left of the apex, heard only during the latter part of each inspiratory act. I could discover no murmur at the base. At a subsequent examination on the same day, made after dinner, the patient having drank a little wine with his dinner, the action of the heart was much greater than at the previous examination. I then discovered a well-marked systolic murmur at the apex, to the left of the apex and at the lower angle of the scapula; I also ascertained the existence of a soft systolic murmur at the base on the left side of the sternum and not on the right side. This murmur extended over the whole summit of the chest on the left side. At the summit it came evidently from the subclavian, as the pitch differed from that of the murmur over the pulmonary artery, i.e., in the 2d intercostal space on the left side.

On the next morning I made an examination while the patient was still in bed. The heart was then acting tranquilly. I discovered a feeble murmur at the apex only; this murmur was not perceived behind, and no murmur was heard at the base.

The aortic and pulmonic second sounds were normal, and so also were the mitral and tricuspid valvular elements of the first sound.

I shall quote from my record book the remarks which were appended to this case when the record was made:—

"The heart is but little if at all enlarged, and the heart sounds are normal. There exist, therefore, no lesions which at present are of serious import. The cardiac trouble which has occasioned the patient so much unhappiness and anxiety, is purely functional.

"Dr. ———— (who first examined this patient) evidently discovered a murmur. His examination was not very critical, and was made after the patient had just mounted stairs at his hotel. The opinion that there was organic disease without any qualifying explanations produced a profound moral impression on the patient. The opinion of Dr. ———— subsequently did something toward revealing the apprehensions of the patient; but his coming such a long distance to consult me is evidence how much his mind was ill at ease on the subject.

"The heart is not entirely free from lesions; there is slight mitral regurgitation. The murmur at the base is perhaps inorganic, or at all events it does not denote important valvular lesions, since a comparison of the aortic and pulmonic sounds show the two to be in a normal relation to each other. The lesions in fact which exist in the case are of no immediate seriousness, and of this I assured the patient in the most positive manner.

"This case affords an illustration of the importance of discriminating between functional disorder and the effects of organic disease when there is evidence of the latter. It illustrates, also, the importance of the heart sounds and of the size of the heart in determining the gravity of lesions. The evils which may arise from the lesions (if they ever occur) are remote, and I felt warranted in assuring the patient that his condition involved no present danger, and that he might dismiss all thoughts of disease of the heart. I ordered him to live well and to resume his out-door sports. His apprehensions were entirely relieved by my assurances, and his expressions of gratification afforded evidence of what he had suffered mentally from the idea of an organic disease incapacitating him from the duties of life and rendering him liable to sudden death."

As I have said, this case is a type of a class of cases of not infrequent occurrence. The existence of a cardiac murmur was discovered in consequence of an examination with reference to the lungs. Prior to this time no symptoms of disorder of the heart existed; the discovery of the murmur was an unfortunate circumstance for the patient; the belief that he had serious disease of the heart became fixed in his mind, and doubtless contributed to the disorder which subsequently occurred. The functional disorder was slight in comparison with cases which are of daily occurrence; but the patient naturally attributed it to organic disease. The affection was in fact altogether functional, albeit the existence of an organic murmur; this is the practical point which the case is intended to illustrate.

I propose now to consider certain practical points pertaining to the cardiac murmur separately; I shall limit my remarks mainly to the murmurs produced by the blood-currents, in the left side of the heart, viz., the *aortic direct, the aortic regurgitant,* the *mitral systolic* and the *mitral direct.* Exclusive of the *pulmonic direct* murmur I have but little practical acquaintance with murmurs emanating from the right side of the heart.

Aortic Direct Murmur.—The question whether a murmur be organic or inorganic has reference generally to a murmur produced by the current of blood from the left ventricle into the aorta. The aortic regurgitant

murmur and a mitral murmur which is truly regurgitant are of necessity organic; they require lesions involving more or less insufficiency of the valves. The mitral direct murmur, as will be seen presently, is inorganic only as a rare exception to the rule. A practical point, then, in certain cases, is to determine whether an existing aortic direct murmur be organic, *i.e.*, dependent on lesions, or whether it be inorganic, *i.e.*, dependent on a blood change. This point cannot always be positively settled, but when such is the case it is practically not very important that it should be settled; in other words, when a murmur exists concerning which we are at a loss to decide whether it be organic or inorganic, if it be the former, the lesion giving rise to it must be trivial, since under these circumstances the heart sounds will be found to be normal and the heart not enlarged. If in connection with an aortic direct murmur we find the aortic second sound impaired and the heart enlarged, we are warranted in considering the murmur organic. But a slight rippling of the current by roughening from an atheromatous or calcareous deposit which occasions no obstruction, and no valvular insufficiency, may yield a murmur. How are we to distinguish this from an inorganic murmur? The absence of the anaemic state, of other cardiac murmurs, of arterial murmurs, of the venous hum, and the persistency and uniformity of the murmur are the circumstances which render it probable that it is organic; while the existence of anaemia, of other cardiac murmurs, of arterial murmurs and the venous hum, together with intermittency and variableness of the murmur, render it probable that it is inorganic.

In my work on diseases of the heart, 1859, I have stated roughness of the murmur to be one of the circumstances showing it to be organic. I then believed that an inorganic murmur was never rough. The able reviewer of my work in the *Dublin Quarterly* says, with regard to this point, "We are unable to give unqualified assent to the statement that an inorganic murmur is uniformly soft." The criticism of the reviewer is just; I was mistaken in the statement as the following case will show:—

I visited in May, 1860, a female patient who presented a loud rasping murmur which had led to the suspicion of aneurism. The patient was exceedingly anaemic; there was total loss of appetite with vomiting and diarrhoea. The anaemia could not be accounted for; it belonged in the category of cases described by Addison as cases of idiopathic anaemia. I found a rough rasping murmur at the base of the heart on the right of the sternum, and a similar murmur was heard over the subclavian and carotid. On examination after death, in this case, the heart was perfectly normal, the aortic orifice, the aorta, subclavians, and carotids were free from any morbid change, and the lungs were healthy. The murmur was evidently due to a blood change.

The discrimination of an aortic direct from a pulmonic direct murmur is a point of interest. If the normal situation of the aortic and pulmonic

artery in relation to the walls of the chest be preserved, an aortic direct murmur has its maximum of intensity and may be limited to the point where the aorta is nearest the surface, viz., the second intercostal space on the right side close to the sternum. But the normal relation of the vessels to the thoracic walls is not infrequently changed when the heart becomes enlarged, or as a consequence of past or present pulmonary disease, and hence this murmur may be loudest or limited to the base on the left side of the sternum. The situation of the murmur or of its maximum, therefore, is not always reliable in the discrimination. A pulmonic direct murmur has its maximum or is limited to the second or third intercostal spaces on the *left* side close to the sternum, the artery being at these points nearest the surface, but, as just stated, an aortic direct murmur may be found to be loudest in this situation. If the heart be not enlarged or displaced by pressure from below the diaphragm, the chest not depressed, and the lungs are free from disease, the fact that a murmur has its maximum at or is limited to the right side of the sternum, is evidence of its being aortic rather than pulmonic, and *per contra*, the fact of a murmur having its maximum at or being limited to the left side of the sternum, is evidence *of its being pulmonic rather than aortic.* But the propagation of the murmur into the carotid is the most important circumstance in this discrimination. An aortic direct murmur, unless it be quite weak, is generally propagated into the carotid. A pulmonic direct murmur of course cannot be. Here attention to the pitch and quality of sound is called into requisition. It is to be determined that a murmur heard over the carotid is propagated from the aorta not produced within the carotid. How is this to be determined? Very easily in most cases, by a simple comparison of the murmur as heard over the carotid and at the aortic orifice. If the murmur in the neck be a propagated murmur it will differ from that at the base of the heart chiefly as regards intensity; the pitch and quality will not be materially changed. If it be rough or soft at the base of the heart, it will be the same in the neck; if the pitch be high or low at the base of the heart, it will be the same in the neck. On the other hand, a murmur produced within the carotid, will be likely, in the great majority of cases to differ in quality and pitch from a coexisting murmur at the aortic orifice.

In accordance with what has been stated with reference to the limitations of the significance of organic murmurs in general, an aortic direct murmur, when undoubtedly organic, alone affords little or no information respecting the nature and extent of the lesions which give rise to it. A comparison of the aortic with the pulmonic second sound of the heart enables us frequently to form an opinion as regards the amount of damage which the aortic valve may have sustained. The aortic second sound, in health, as heard in the right second intercostal space near the sternum, is more intense, and has a more marked valvular quality, than

the pulmonic second sound as heard in a corresponding situation on the left side. Now, it is often easy to determine whether the intensity of the aortic second sound is diminished and its valvular quality impaired; and in proportion as this sound is abnormally altered in these respects, we may infer that the aortic valve is damaged. It is hardly necessary to say that, in order for this comparison to warrant the inference just stated, pulmonary disease must be excluded. A tuberculous deposit, for example, on the left side, may, by conduction, render the pulmonic apparently more intense than the aortic sound, the latter retaining its normal intensity; the same will occur from shrinking of the upper lobe of the left lung so as to bring the pulmonary artery into contact with the thoracic walls. Under the latter circumstances the pulsation of the pulmonic artery may sometimes be distinctly felt in the second left inter-costal space near the sternum. I have met with two cases during the past winter in which the pulsation of the pulmonic artery was so strong as to suggest the idea of aneurism; in both cases the patients were affected with tuberculous disease of the left lung. Alteration of the normal relation of the aorta and pulmonic artery due to enlargement of the heart, or to any of the causes already mentioned, will of course preclude a comparison of the two sounds.

With reference to the value of a comparison of the aortic and pulmonic second sound in estimating the amount of aortic lesions, the able reviewer in the *Dublin Quarterly,* to whom I have already referred, and for whose valuable criticisms I beg to avail myself of this opportunity of expressing my sincere thanks, remarks as follows:—

"It is observed, to our great wonder, that if the aortic second sound retain its normal intensity and purity, it shows that the aortic valve is competent to fulfil its function, *a fact which warrants the exclusion of lesions affecting it sufficiently to give rise to obstruction."* He adds, "Surely Dr. Flint must have become clinically cognizant of the fact that there is not unfrequently serious contraction of the aortic orifice producing marked obstruction and hypertrophy of the left ventricle, the aortic second sound remaining intact."

This criticism is not altogether just. I state that the normal intensity and purity of the aortic second sound warrant the exclusion of lesions affecting it, *i.e.,* the valve, sufficiently to give rise to obstruction. I do not say that contraction of the aortic orifice may not occur without involving the aortic valve, and, in such a case, the aortic second sound may remain intact. In fact, I imply this when I proceed to say, "In a large proportion of the cases of obstructive lesions of the aortic orifice, the valve is involved sufficiently to compromise, to a greater or less extent, its function and impair the intensity of the aortic second sound." This language is equivalent to admitting that there is a small proportion of cases of obstructive lesions of the aortic orifice, in which the valve is *not* involved

sufficiently to compromise its function and impair the intensity of the aortic second sound. These exceptional cases are extremely rare. Surely the able reviewer will admit that, in the great majority of cases, the valve is involved so as to impair its function to a greater or less extent.

I have lately been interested in a nice point of observation connected with the murmur under consideration, viz., the concurrence of two aortic direct murmurs, one produced at the aortic orifice and another within the aorta just above the orifice. One of the murmurs may be organic and the other inorganic, or both murmurs may be organic. At the present moment I have under observation three cases of endocarditis with rheumatism, each presenting a high pitched basic murmur when the stethoscope is placed over the sternum and a little to the right of the median line, the murmur limited to a circumscribed space, and just above this point, in the right second intercostal space, is another murmur differing from the former notably in pitch, being quite low. In one of these cases there is still another murmur in the pulmonic artery. The high pitched murmur just below the second intercostal space, as I infer from the situation to which it is limited, is a murmur produced at the aortic orifice; and the low pitched murmur just above, as I infer, also, from the situation to which it is limited, is an aortic murmur produced within the artery above the aortic orifice. I infer that there are two murmurs from the notable difference in pitch, it being by no means probable that a single sound would be so much altered within the area in which the two murmurs are heard, this area not being larger than a half dollar. That an aortic murmur is sometimes produced at the orifice and sometimes within the artery above the orifice, in different cases, is certain, but I am not aware that the production of a murmur in each situation, at the same time, in the same case, and the discrimination of the two by means of the character of the sound, have been pointed out.

Aortic Regurgitant Murmur.—This murmur need never, as a matter of course, be confounded with the systolic murmurs, viz., the aortic direct, and mitral regurgitant, the latter occurring with the first, and the former with the second sound of the heart. In general, too, there is no difficulty in distinguishing the aortic regurgitant, from the mitral direct murmur. The former occurs with and follows the second sound, the latter precedes the first sound. The one is diastolic, the other is pre-systolic. This is a distinction, nice, it is true, but easily appreciable in practice, to which I shall recur under the heading of the mitral direct murmur.

The situation of the murmur is also distinctive. It is best heard at, and below the base of the heart. Usually it is best heard below the base to the left of the median line on a level with the third or fourth ribs. This is doubted by the reviewer in the *Dublin Quarterly*, to whom I have referred, but as the statement is based on a pretty large number of recorded observations, I must consider it as correct. It is not uncommon to hear

this murmur distinctly, and even loudly, over the apex; it may be diffused over the whole praecordia and even propagated beyond this region.

An aortic murmur with the second sound of the heart, propagated below the base of the heart, necessarily implies regurgitation, in other words there must be insufficiency of the aortic valvular segments. But it is always to be borne in mind that no inference can be drawn from the intensity or character of the murmur, respecting the amount of insufficiency and consequent regurgitation. An extremely small regurgitant stream may give rise to a loud murmur, while a feeble murmur may accompany a large regurgitant current, as the rippling brook is noisy while the deep broad river flows silently. In a case recently under observation, there existed a loud aortic regurgitant murmur, and on examination after death the aortic segments were so slightly impaired that, on cursory inspection, they might have been considered as normal. Weakening or extinction of the aortic second sound of the heart are points of importance as showing frequently the extent to which the function of the aortic valve is impaired. Comparison with the pulmonic sound enables us to judge whether the aortic sound be impaired, provided the pulmonic sound be not abnormally intensified as a result of coexisting mitral lesions. It is important to recollect that when aortic and mitral lesions coexist, the intensity of the pulmonic sound cannot be taken as a criterion for judging whether the aortic sound be, or be not weakened. This remark is equally applicable to the comparison in cases in which an aortic direct murmur is present. It is needless to say that in comparing the aortic and pulmonic sound in connection with an aortic regurgitant, as with an aortic direct murmur, pulmonary disease is to be excluded, *i.e.,* solidification or shrinking of the left lung will, as already stated, render the pulmonic sound relatively more intense than the aortic, irrespective of, on the one hand, any actual increase of the intensity of that sound, or, on the other hand, of any weakening of the aortic sound. It is also to be stated here, as heretofore, that an alteration of the situation of the aorta and pulmonary artery as regards the thoracic walls, due to enlargement of the heart, or other causes, will preclude a comparison of the two sounds with reference either to intensification of the pulmonic, or weakening of the aortic sound.

Mitral Systolic Murmur.—I use the phrase mitral *systolic,* instead of that more commonly used, viz., mitral *regurgitant* murmur, as applied to any murmur produced at the mitral orifice and accompanying the first sound of the heart. If the latter term be applied to any systolic murmur emanating from the mitral orifice, we fall into the solecism of calling a murmur regurgitant in cases in which there is no regurgitation. A mitral murmur may be produced by mere roughness of the valvular curtains when there is no insufficiency of the valve. In this case the murmur cannot be correctly said to be regurgitant. A mitral systolic murmur, thus, may or may not be a regurgitant murmur, and, to express this important

distinction, we may say that a mitral systolic murmur exists with or without regurgitation. The question at once arises, how are we to determine whether a mitral systolic murmur be regurgitant or non-regurgitant? This point claims consideration.

A mitral systolic murmur, as is well known, generally has its maximum of intensity at, and the murmuring may be limited to, the situation of the apex-beat, or to the point where the intensity of the first sound of the heart is greatest. The murmur may be diffused, in the first place, within this point over the body of the heart, and, in the second place, without the apex over the left lateral surface of the chest and on the back. I have been led to believe that when the murmur is diffused over the left lateral surface and more or less over the back, it always denotes regurgitation, and that when the murmur is not propagated much without the apex, although it may be more or less diffused over the body of the heart, it may be produced within the ventricle and not by a regurgitant current. In the latter case I have distinguished the murmur as an intra-ventricular murmur, and not considered it as affording any evidence of insufficiency of the mitral valve. It is this intra-ventricular, or mitral systolic non-regurgitant murmur, which generally exists in rheumatic endocarditis. The importance of the point involved is obvious, for a murmur emanating from the mitral orifice without valvular insufficiency or regurgitation, denotes lesions of little immediate consequence, and they may be innocuous, not only for the present but for the future.

The practical rule just stated, I believe, generally holds good; but there may be exceptions. The following is perhaps an exceptional instance: A case was recently under my observation in Bellevue Hospital, in which acute rheumatism was complicated with endocarditis, pericarditis, and pleurisy, with considerable effusion, affecting the left side. The patient presented, on admission, a loud pericardial friction sound diffused over the whole praecordia, and a loud mitral systolic murmur. The latter had its maximum of intensity at the apex, but was diffused over the left lateral surface of the chest and heard on the back. After the lapse of about a week the friction sound disappeared; but before the disappearance of the friction sound, the endocardial murmur had gradually diminished and disappeared. The pleuritic effusion also disappeared, and evidence was afforded in this case of pericardial adhesions by the immobility of the apex-beat when the body of the patient was placed in different positions. The disappearance of an endocardial murmur developed by rheumatic endocarditis, so far as my observation goes, is rare, although I have met with other examples. I suppose that endocarditis does not involve actual regurgitation save as a remote consequence of lesions to which the endocarditis may give rise. I may be mistaken in this supposition, but, assuming that I am not, here was an instance in which

an intra-ventricular or non-regurgitant mitral systolic murmur was propagated entirely around the chest.

With reference to determining the existence of either regurgitation or obstruction, or both, resulting from mitral lesions, a comparison of the aortic and pulmonic second sound, forms a beautiful and useful application of auscultation. Obstructive and regurgitant lesions, situated at the mitral orifice, involving an obstacle to the free passage of blood through the pulmonary circuit, give rise, as is well known, to hypertrophy of the right ventricle. In this way they lead to intensification of the pulmonic second sound of the heart. This effect is due, in part, to the augmented power of the contractions of the right ventricle, and, in part, to the resistance to the passage of blood through the lungs, both continuing to increase the dilatation of the pulmonary artery by the pulmonic direct current, and the consequent recoil of the arterial coats by which the pulmonic valvular segments are expanded, and the pulmonic second sound produced. But the morbid disparity between the aortic and pulmonic second sound is due, not alone to the intensification of the latter in the manner just stated. The aortic second sound is weakened in proportion to the amount of blood which fails to pass into the aorta with the ventricular systole, in consequence of the mitral obstruction or regurgitation. It is obvious that the aortic direct current will be lessened by the amount of blood which, in consequence of valvular insufficiency, flows backward into the left auricle after the ventricle contracts, and by the amount of difficulty which exists in the free passage of blood from the auricle into the ventricle in consequence of a contracted orifice. It is also obvious that, other things being equal, the intensity of the aortic second sound will be greater or less according to the quantity of blood propelled into the aorta by the ventricular systole. Thus, it is clear how mitral obstruction and regurgitation lead to weakening of the aortic sound, as well as to intensification of the pulmonic sound, and both effects are abundantly attested by clinical observation.

The degree of weakening of the aortic and of intensification of the pulmonic sound will be proportionate to the amount of mitral regurgitation or obstruction, or both. We have then, in this application of auscultation, a means of obtaining information respecting the extent or gravity of mitral lesions. And, in a negative point of view, this application is important, viz., as a means of determining that lesions which give rise to a murmur are not serious; in other words, of determining that they do not involve much, if any, obstruction or regurgitation. As enabling us to exclude obstructive or regurgitant lesions in certain of the cases in which mitral murmurs exist, a comparison of the aortic and pulmonic sound is of great practical value. But the circumstances which may stand in the way of this application of auscultation are to be borne in

mind. The two sounds cannot be compared with reference to mitral, more than with reference to aortic lesions, if there be coexisting pulmonary disease, nor whenever the normal relation of the aorta and pulmonary artery to the thoracic walls is altered by either past or present disease of the lungs, by deformity of the chest, or any other cause. It is also to be recollected that mere enlargement of the heart may disturb the normal relation of these vessels to the walls of the chest. This application, moreover, cannot be made when mitral and aortic lesions coexist. Under the latter circumstances it is, of course, difficult or impossible to determine how far an existing disparity between the aortic and pulmonic sound is due to the aortic, and how far to the mitral lesions.

Another important point pertaining to a mitral systolic murmur is, its occurrence without any appreciable lesions. A truly mitral regurgitant murmur doubtless always involves lesions of some kind, for it is hardly probable that the papillary muscles, as has been supposed, may become spasmodically affected and thus give rise to insufficiency or regurgitation as a temporary functional disorder. But it is undoubtedly true that a systolic murmur either limited to, or having its maximum of intensity near the apex, has been repeatedly observed in cases in which mitral lesions were not apparent after death. Dr. Bristowe in a paper contained in the *Brit. and For. Med. Chir. Review*, for July, 1861, details six cases of this description. Dr. Barlow, in an article in *Guy's Hospital Reports*, vol. v., 1859, states that a mitral murmur may occur (for what reason he does not state) in long-continued capillary bronchitis. I have met with some instances in which a systolic murmur, supposed to be mitral, existed, and no mitral lesions were found after death.

Case 1.[1] In the winter of 1859-60, I saw a female patient in the Charity Hospital, New Orleans, in the service of my colleague, Prof. Brickell, affected with capillary bronchitis. After several days there was improvement as regards the pulmonary symptoms, and then, for the first time, a systolic cardiac murmur was discovered. The murmur was loudest at the epigastrium, but heard over the site of the apex, and extended to, but not above the base of the heart. The patient subsequently died. On examination after death the lungs were emphysematous; there were *no* valvular lesions, all the valves appearing to be sound. The foramen ovale was closed. There were no clots. The right ventricle was distended with liquid blood. The walls of the heart were of normal thickness. The valves and orifices were not measured, nor was the water test of valvular sufficiency employed.

In recording this case I have commented on the murmur as follows: "What could have caused the loud systolic murmur? I cannot say unless it was due to distension of the right ventricle and tricuspid regurgitation."

In support of the supposition that the murmur was tricuspid, not mitral, it is to be noted that the greatest intensity was at the epigastrium. It was, however, considered to be a mitral systolic murmur during life.

Case 2.[1] During the winter of 1860-61, a patient was under my observation in the Charity Hospital, New Orleans, for four months, affected with albuminuria and general dropsy. During all this time there was a mitral systolic murmur at the apex and over the body of the heart, and not propagated without the apex. It was regarded as a mitral systolic, nonregurgitant or intra-ventricular murmur, and as such pointed out to several private classes in auscultation. The patient died by asthenia, and was found to have fatty kidneys and cirrhosis of the liver. On examination of the heart, *post mortem*, nothing abnormal was found except some enlargement, the organ weighing 12 oz., and a little separation of the marginal extremity of two of the aortic segments. The mitral valve appeared to be perfectly normal. I expected to find some roughening of the mitral valve but no insufficiency; there was, however, no atheromatous, calcareous or other deposit, and the valve seemed to be sufficient. There was no aortic, nor pulmonic murmur in this case, a fact which excludes the supposition that the existing murmur was due to the condition of the blood.

Case 3.[2] During the winter of 1860-61, a patient was under my observation in the Charity Hospital, New Orleans, for about six weeks, affected with chronic bronchitis and emphysema of lungs. He presented habitual dyspnoea which was at times excessive, persisting lividity and anasarca. The heart was evidently somewhat enlarged. There was a loud rough systolic murmur, having its maximum of intensity at the apex propagated without the apex (the record does not state how far), and over the body of the heart. On examination after death the volume of the heart was not much increased, and its weight was 13 oz. The left ventricle was not dilated and the left auricle was small. The walls of the left ventricle did not exceed half an inch in thickness, and the appearance of the muscular tissue was healthy. The mitral valve was perfectly normal. The orifice was not enlarged, and the valve must have been sufficient. No lesion at the aortic orifice. The right cavities were much dilated. They were twice as large as the left cavities. The walls of the right ventricle were much thickened, the thickness falling but little short of that of the left ventricle. No lesion of the pulmonic orifice. The tricuspid valve was normal. The orifice was very large, admitting the extremities of all the fingers. I have appended to the record of this case the following comment. "Whence the murmur supposed to be a mitral regurgitant? I suspect it was a tricuspid regurgitant."

Dr. Bristowe, in the article already referred to, discusses several conditions which have been supposed to give rise to the murmur in cases like those which have just been given, viz., clots in the ventricular cavity,

[1]Private Records, vol. xi. p. 243.
[2]Hospital Records, vol. xv. p. 423.

spasm of the papillary muscles, and enlargement of the auricular orifice so as to render the valve insufficient. His own opinion is that the murmur is due to a "disproportion between the size of the ventricular cavity and the length of the chordae tendineae and musculi papillares." This disproportion he attributes to dilatation of the cavity of the ventricle. He also accepts to some extent an explanation offered by Dr. Hare, viz., that the murmur may be due to a "lateral displacement of the origins of the musculi papillares in consequence of the rounded form which dilatation imparts to the heart."

These several explanations may each be applicable to certain cases, but none of them, apparently, to the cases which I have given. Clots in the left ventricular cavity were not present in either of the cases; the murmur continued too long and too persistently to be due to spasm; the mitral orifice was not dilated, and the enlargement of the heart was not sufficient to occasion a notable disproportion between the length of the tendinous cords and papillary muscles, and the ventricular cavity. I am disposed to think that in each of the three cases the murmur was erroneously considered to be mitral, that it was a tricuspid regurgitant murmur. As I have already said, I have but little practical knowledge of tricuspid murmurs. I have met with two instances in which murmur was connected with well-marked tricuspid lesions as verified by examination after death. In both these cases the murmur was heard over the body of the heart, within the superficial cardiac region. I suspect that a tricuspid regurgitant murmur is not so rare as is generally supposed, and that not very infrequently it is considered to be mitral. This opinion is expressed by Dr. Gairdner in an interesting article on cardiac murmurs in the *Edinburgh Med. Monthly*, Nov., 1861. According to this able clinical observer, a tricuspid systolic murmur is heard over the right ventricle where it is uncovered of lung, being but slightly audible above the third rib; and, if the heart be much enlarged, it may be heard louder towards the xiphoid cartilage. A collection of clinical facts respecting the frequency of tricuspid murmurs, the physical conditions giving rise to them, and the means of discriminating them from mitral murmurs, is an important desideratum.

Mitral Direct Murmur.—This murmur is not recognized by many auscultators, and its existence is denied by some. It is generally confounded with a mitral systolic murmur. For many years after I had begun to devote special attention to cardiac affections, I committed this mistake, and I was sometimes puzzled to account for a supposed mitral systolic murmur rough at its beginning and soft at its ending. In my records of some cases before I had learned to separate the mitral direct from a mitral regurgitant, I have described the latter as presenting the variation just stated, the fact being that the two murmurs were present, the one rough and the other soft. It is only within the last few years that I have discriminated

these two murmurs, but during this time my field of clinical observation has been so extensive that I have had abundant opportunities to make the discrimination. With regard to the frequency of the mitral direct murmur, it is by no means so rare as is generally supposed, and as I had thought some years ago. At one time during the past winter, in Bellevue Hospital, I knew of six examples of it, and several also at the Blackwell's Island Hospital. When the auscultator has learned to distinguish it, he will not be long in finding it if he be in the way of seeing a moderate number of cases of disease of the heart. From what has now been said, it is obvious that an important point pertaining to this murmur is, its discrimination from other murmurs. This point will first claim consideration.

In order to comprehend this murmur, it is essential to understand clearly when the mitral direct current of blood takes place. The opportunity of observing the movements of the heart exposed to view in a living animal, conduces greatly to a clear understanding of this point. The mitral direct current is produced by the contraction of the auricles; now, when do the auricles contract? When the movements of the heart are observed, it is seen that the contraction of the auricles immediately precedes the contraction of the ventricles. So close is the connection between the contraction of the auricles and the contraction of the ventricles, that the former appears to merge into the latter; there is no appreciable interval between the two, but the successive movements, although distinct, appear to be continuous. Moreover, it is evident to the eye, and to the touch, that the contractions of the auricles are not so feeble as some seem to suppose. The mitral direct current of blood, therefore, occurs just before the ventricular systole; it continues up to the ventricular systole, and must, of course, instantly cease when the ventricles contract. The contraction of the ventricles causing the first sound of the heart, it follows that the mitral direct current caused by the auricular contractions must take place just before the first sound; that it must continue to the first sound, and that it cannot continue an instant after the first sound.

The mitral direct murmur is produced by the mitral direct current of blood forced by the auricular contractions through a contracted or roughened mitral orifice. Hence, the facts just stated with regard to the current, apply to the murmur. The murmur occurs just before the ventricular systole or the first sound of the heart; it continues up to the occurrence of the first sound, and instantly ceases when the first sound is heard. It is not strictly correct to call this a diastolic murmur; it does not accompany the second or diastolic sound of the heart. The aortic regurgitant is the only true diastolic murmur. The mitral direct is a pre-systolic murmur; this name expresses its proper relation to the heart sounds, and it is the only murmur which does occur in that particular

relation. The time of its occurrence as just explained, and as expressed by the term pre-systolic, is sufficient for its easy recognition when once it is fully comprehended. Although, when this murmur is fully comprehended, and has been repeatedly verified, it is more readily recognized than either of the other murmurs, there is often at first considerable difficulty in determining its existence. Let me endeavour to point out the way in which it may be ascertained. I have already said that by those who overlook this murmur it is generally confounded with the mitral systolic or regurgitant murmur. This is in consequence of its close connection with the first sound, and because it is heard at and near the apex of the heart. Now it is evident that a mitral systolic murmur cannot commence before the ventricular systole. It is equally evident that the ventricular systole and the first sound of the heart are synchronous. It is, therefore, an absurdity to suppose that a mitral systolic or regurgitant murmur can be pre-systolic in the time of its occurrence. This murmur must necessarily accompany and follow the first sound of the heart, as clinical observation has established. We have, then, only to determine that a murmur is pre-systolic, and that it does not accompany the second sound of the heart (i.e., there is an appreciable interval of time between the second sound and the murmur), to recognize it as a mitral direct murmur. Generally it is sufficiently easy, after a little practice, to perceive that the murmur precedes the sound, but, if there be difficulty or doubt, there is a ready mode of rendering it apparent; this is by placing the finger on the carotid pulse. The carotid pulse is synchronous with the first sound of the heart, or, at least, so nearly synchronous, that there is no appreciable interval of time between them. Placing, then, the finger on the carotid and listening to the murmur at the apex, the murmur is found to occur before the arterial impulse and to cease instantly when the latter is felt.

The mitral direct murmur is to be discriminated from an aortic regurgitant murmur. These two murmurs may be confounded at first, but after a little practice the discrimination is easy. The aortic regurgitant murmur accompanies and follows the second sound of the heart, the mitral direct commences after the second sound. Generally there is a distinctly appreciable interval of time between the second sound and the commencement of the murmur. The aortic regurgitant murmur may be prolonged nearly or quite through the long pause up to the first sound; but the intensity of the murmur diminishes with the prolongation, the murmur being insensibly lost before or when the first sound occurs. The mitral direct murmur, on the contrary, always continues up to the first sound, and instead of losing any of its intensity, it becomes more intense, and appears to be abruptly arrested, in its greatest intensity, when the first sound occurs. This is a striking characteristic. The difference in the situation in which two murmurs respectively are heard with their

maximum of intensity, is another point in the discrimination. The aortic regurgitant murmur is generally heard at the base of the heart, and is heard loudest a little below the base near the left margin of the sternum on a level with the third intercostal space. The mitral direct murmur is heard loudest at or a little within the apex; is generally confined within a circumscribed space, not propagated much without the apex and rarely to the base of the heart.

The quality of the mitral direct murmur is, in many cases, characteristic. In my work on diseases of the heart I have said that this murmur is generally soft. My experience since that work was written has shown me that this statement is incorrect. The murmur is oftener rough than soft. The roughness is often peculiar. It is a *blubbering* sound, resembling that produced by throwing the lips or the tongue into vibration with the breath in expiration. I suppose that the murmur is caused, in these cases, by the vibration of the mitral curtains, and that the vibration of the lips or tongue by the breath represents the mechanism of the murmur as well as imitates the character of the sound. At one time I supposed this blubbering murmur denoted a particular lesion, viz., adhesion of the mitral curtains at their sides, forming that species of mitral contraction known as the *buttonhole slit;* but I have found this variety of murmur to occur without that lesion, and, in fact, as will be seen presently, when no mitral lesion whatever exists.

A mitral direct murmur may, or may not, be associated with a mitral systolic murmur. Without having analyzed the numerous examples which I have recorded during the last few years, I should say that, while the mitral systolic murmur is much more frequent in its occurrence than the mitral direct, the former, indeed, being the most common of all the murmurs, the mitral direct is observed quite as often without, as with the mitral systolic. But the two frequently coexist, and then the demonstration of the existence of the mitral direct murmur may be made more striking than when it exists alone, provided, as is usually the case, this murmur be rough and the mitral systolic murmur be soft. Listening at or near the apex in a case presenting a blubbering mitral direct and a soft mitral systolic murmur, the former, of course, precedes the latter, and between the two occurs the first sound of the heart, the apex-beat and the carotid pulse. The first sound, the apex beat or the carotid pulse will be found to mark the abrupt ending of the mitral direct, and the beginning of the mitral systolic murmur. The different relations of the two murmurs to the first sound are distinctly perceived in such a case if the observer be prepared to perceive them by a clear comprehension of the subject. And when once the discrimination between the two murmurs has been fairly made, it becomes sufficiently easy; indeed, the mitral direct murmur is then more readily recognized than either of the other murmurs.

The existence of a mitral direct murmur has been theoretically denied on the ground that the auricular contractions are too weak to propel the current of blood with sufficient force to give rise to a sound. It is undoubtedly true that, other things being equal, the intensity of a murmur is proportionate to the force of the current, and clinical observation shows that sometimes a murmur is not appreciable when the heart is acting feebly, but becomes distinct when the power of the heart's action is from any cause increased. But murmurs do by no means always require for the production a powerful action of the heart; on the contrary, loud murmurs are often found when the heart is acting very feebly. For example, I have reported a case in which an aortic direct and an aortic regurgitant murmur were well marked in a patient an hour before death, the patient dying from paralysis of the heart due to distension of the left ventricle. Venous murmurs in the neck are often notably loud when, assuredly, the force of the current of blood in these veins is vastly less than the current from the auricles to the ventricles. The feebleness of the current in this instance is shown by the slight pressure requisite to interrupt it and arrest the murmur. It requires but little force of the expiratory current of air to throw the lips into vibration so as to produce a loud sound. Moreover, one has only to see and feel the contractions of the auricle, when the heart is exposed in a living animal (the heart's action being much weakened under these circumstances) to be convinced that the power of these contractions is not so small as some seem to imagine; the blood is driven into the ventricles with considerable force. It is hardly necessary to say, however, that *a priori* reasoning with regard to the existence or non-existence of physical signs is not admissible. Their existence is a matter to be determined by direct observation. Clinical observation shows that a murmur does occur at the precise time when the mitral direct current takes place as shown by observation of the movements of the heart exposed to view in a living animal. And clinical observation shows that this murmur is not always feeble, but, on the contrary, is not infrequently notably loud.

So much for the reality of the mitral direct murmur and the means of discriminating it from other murmurs. It remains to consider another important practical point, viz., the pathological import of this murmur. As already stated, it is developed in connection with a contracted mitral orifice and, so far as my experience goes, especially in connection with contraction caused by adherence of the mitral curtains, forming the *buttonhole slit;* the murmur, then, being due, not to the passage of blood over a roughened surface, but to vibration of the curtains. And the sound, as thus produced, is peculiar, resembling the sound which may be produced, in an analogous manner, by causing the lips to vibrate with an expiratory puff. The murmur, however, may be produced by the flowing of the current of blood over a roughened surface, without contraction of the aperture. This

is undoubtedly rare. As a rule, the force of the mitral direct current is not sufficient to develop a murmur unless there be mitral contraction. Is this murmur ever produced without any mitral lesions? One would *a priori* suppose the answer to this question to be in the negative. Clinical observation, however, shows that the question is to be answered in the affirmative. I have met with two cases in which a well-marked mitral direct murmur existed, and after death in one of the cases no mitral lesions were found; in the other case the lesion was insignificant. I will proceed to give an account of these cases, and then endeavour to explain the occurrence of the murmur.

Case 1.[1] In May, 1860, I examined a patient, aged 56, who had had repeated attacks of palpitation, sense of suffocation, with expectoration of bloody mucus and a feeling of impending dissolution, but without pain, the paroxysms resembling angina, excepting the absence of pain. In the intervals between these attacks he was free from palpitation, did not suffer from want of breath on active exercise, and considered himself in good health. He had never had rheumatism. On examination of the chest, the heart was found to be enlarged, the enlargement being evidently by hypertrophy. At the apex was a pre-systolic blubbering murmur, which I then supposed to be characteristic of the buttonhole contraction of the mitral orifice. At the base of the heart was an aortic regurgitant murmur, which was diffused over nearly the whole praecordia. There was no systolic murmur at the base or apex. Three days after this examination the patient was attacked with another paroxysm; and died in a few moments after the attack, sitting in his chair. The heart was enlarged, weighing 16½ oz., the walls of the left ventricle measuring four-fifths of an inch. The aorta was atheromatous, and dilated so as to render the valvular segments evidently insufficient. The mitral valve presented nothing abnormal, save a few small vegetations at the base of the curtains, as seen from the auricular aspect of the orifice.

In this case it is assumed that the mitral direct murmur, which was loud and of the blubbering character, was not due to the minute vegetations which were found after death. There was no mitral contraction. The mitral valve was unimpaired, so that the murmur could not have been due to mitral regurgitation.

Case 2.[2] In February, 1861, I was requested to determine the murmur in a case at the Charity Hospital, New Orleans. I found an aortic direct and an aortic regurgitant murmur, both murmurs being well marked. There was also a distinct pre-systolic murmur within the apex, having the blubbering character. On examination after death, the aorta was dilated and roughened with atheroma and calcareous deposit. The aortic segments were contracted, and evidently insufficient. The mitral curtains presented no lesions; the mitral orifice was neither contracted nor dilated,

[1]Private Records, vol. x. p. 713
[2]Ibid., vol. xi. p. 241.

and the valve was evidently sufficient. The heart was considerably enlarged, weighing 17½ oz., and the walls of the left ventricle were an inch in thickness.

In the second, as in the first of the foregoing cases, it is evident that a mitral systolic murmur was not mistaken for a mitral direct murmur, for in both cases, the conditions for a mitral systolic murmur were not present. In both cases the mitral direct murmur was loud and had that character of sound which I suppose to be due to vibration of the mitral curtains. In both cases, it will be observed, an aortic regurgitant murmur existed, and aortic insufficiency was found to exist post mortem. How is the occurrence of the mitral direct murmur in these cases to be explained? I shall give an explanation which is to my mind satisfactory.

The explanation involves a point connected with the physiological action of the auricular valves. Experiments show that when the ventricles are filled with a liquid, the valvular curtains are floated away from the ventricular sides, approximating to each other and tending to closure of the auricular orifice. In fact, as first shown by Drs. Baumgarten and Hamernik, of Germany, a forcible injection of liquid into the left ventricle through the auricular opening will cause a complete closure of this opening by the coaptation of the mitral curtains, so that these authors contend that the natural closure of the auricular orifices is effected, not by contraction of the ventricles, but by the forcible current of blood propelled into the ventricles by the auricles. However this may be, that the mitral curtains are floated out and brought into apposition to each other by simply distending the ventricular cavity with liquid, is a fact sufficiently established and easily verified. Now in cases of considerable aortic insufficiency, the left ventricle is rapidly filled with blood flowing back from the aorta as well as from the auricle, before the auricular contraction takes place. The distension of the ventricle is such that the mitral curtains are brought into coaptation, and when the auricular contraction takes place the mitral direct current passing between the curtains throws them into vibration and gives rise to the characteristic blubbering murmur. The physical condition is in effect analogous to contraction of the mitral orifice from an adhesion of the curtains at their sides, the latter condition, as clinical observation abundantly proves, giving rise to a mitral direct murmur of a similar character.

A mitral direct murmur, then, may exist without mitral contraction and without any mitral lesions, provided there be aortic lesions involving considerable aortic regurgitation. This murmur by no means accompanies aortic regurgitant lesions as a rule; we meet with an aortic regurgitant murmur frequently when not accompanied by the mitral direct murmur. The circumstances which may be required to develop, functionally, the latter murmur, in addition to the amount of aortic regurgitation, remain to be ascertained. Probably enlargement of the left ventricle is one

condition. The practical conclusion to be drawn from the two cases which have been given is, that a mitral direct murmur in a case presenting an aortic regurgitant murmur and cardiac enlargement, is not positive proof of the existence of mitral contraction or of any mitral lesions. The coexistence of a murmur denoting mitral regurgitation, in such a case, should be considered as rendering it probable that the mitral direct murmur is due to contraction or other lesions, and not functional.

Dr. Gairdner, in a recent article already referred to, proposes a change of name for the mitral direct murmur. He proposes to call it an auricular systolic murmur. Inasmuch as the murmur is produced by the systole of the left auricle, this name is significant. And the usual name is open to this criticism, viz.; it is not produced by the whole of the mitral direct current, but only that part of the current which is caused by the contraction or systole of the auricle. From the situation of the auricles as regards the ventricles, the former being placed above the latter, and the free communication by means of the auriculo-ventricular openings, the blood must begin to flow from the auricles into the ventricles the instant the ventricular contractions cease. During the first part of the long pause or interval of silence, *i.e.,* the period after the second sound and before the subsequent first sound of the heart, the blood flows from the auricles into the ventricles simply in obedience to gravitation. It is not ascertained that this part of the current ever gives rise to a murmur. If it does, the murmur would follow immediately the second sound, or when an aortic regurgitant murmur occurs. I have conjectured that such a mitral direct murmur may occur, and that it is confounded with an aortic regurgitant murmur. This conjecture is based on cases in which an apparent aortic regurgitant murmur existed, and the aortic valves seemed to be nearly or quite sufficient on examination after death. However this may be, the mitral direct current giving rise to the murmur which has been considered in this article, is not the current which immediately follows the second sound, and is due to gravitation alone, but it is the current immediately preceding the ventricular systole, and due to the systole of the auricle. Hence, as it seems to me, the name proposed by Dr. Gairdner, being more specific and accurate, is to be preferred to that in common use.
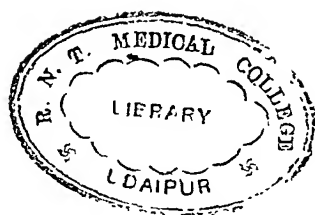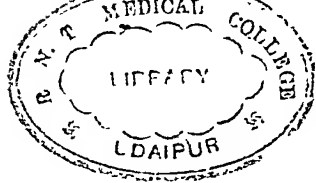
1867

PIERRE CARL ÉDOUARD POTAIN
DESCRIPTION OF THE MOVEMENTS AND THE
MURMURS OF THE JUGULAR VEINS

PIERRE CARL E POTAIN

PIERRE CARL EDOUARD POTAIN

(1825-1901)

$\mathscr{P}$IERRE CARL EDOUARD POTAIN, who was born in Paris on July 19, 1825, came from a long line of physicians: indeed, one of his ancestors is known to have practiced surgery in 1662. Potain, himself, used to say, "Without a doubt, there must have been some barber at the origin [of the family], but it was in a time too remote to be found now."

Potain's father was postmaster of Saint-Germain and set his heart on his son's returning to the family tradition of medicine. When Potain was very young, his father took him, at the close of day, to the forest of Saint-Germain and taught him all that he knew of the natural sciences, grammar and literature. He learned the German language from his mother.

Potain could not afford a college education, but he studied by himself and soon was able to pass the examinations at the University of Paris, where he received the degree of Bachelor of Arts. His own tastes inclined him to science and mechanics, but urged by his father, he began the study of medicine, and in 1848 passed the examinations which permitted him to serve an internship at the hospitals in Paris. In 1849, while at the famous Salpêtrière Hospital, he suffered an attack of cholera, but soon recovered from it. While convalescing at Metz he suffered a second attack of cholera, but managed to survive it.

After he received the degree of Doctor of Medicine in 1853 from the University of Paris he decided to accept a position as assistant to Jules Gabriel François Baillarger (1806-1891) at the asylum for the insane at Ivry. In 1856 he returned to Paris, where he worked under Jean-Baptiste Bouillaud (1796-1881), who was the first to describe "gallop-rhythm." Potain became chief of Bouillaud's clinic and later served on the staffs of the Hôpital Sainte-Antoine and the Hôpital Necker. In 1859 he became an assistant professor at the medical school of the University of Paris. In 1861 he took the competitive examination and was named physician of the Hospitals of Paris and associate professor of the Faculty of Medicine of Paris.

In 1870, during the war, Potain was asked to take charge of an ambulance company, but he decided, instead, to enlist in the regular army as a common "carabinier." When not on duty he returned to the hospitals to care for the wounded. In 1876 he was appointed professor of pathology at the University of Paris. Later that year he was transferred to the chair of clinical medicine. In 1882 he became associated with the Charity Hospital in Paris, where he remained until the age of retirement.

In 1883 Potain was elected to the Académie de Médecine, and in 1893, to the Académie des Sciences, the Institute of France. He was elected a commander of the Legion of Honor.

Potain numbered among his patients Henri C. F. M. Dieudonné, Comte de Chambord (1820-1883). On one occasion when he received a call from the count, who was the Pretender to the throne of France, he sent his friend, Edmé Félix Alfred Vulpian (1826-1887), to see the count while he stayed to comfort an ill friend, Dr. Parrot, for whom he knew very well he could do nothing.

[531]

In 1900 Potain reached the age of retirement. He was sorry to leave. Louis Henri Vaquez (1860-1936), his former pupil and Louis Joseph Teissier (1851-1926), his assistant, went to wish him a Happy New Year on January 1, 1901, and told him to come back to do research. To this Potain replied: "No, you see, it is well over; when the function is through, the organ must disappear."

He died suddenly within the week, and was buried on January 8, 1901.

Potain realized the importance of recording the heart beats in connection with the venous pulsations and the graph of the pulse, and he devised an instrument with which to make these recordings possible.

His chief contributions to medicine included: (1) making precise the relationship of tricuspid regurgitation and circulatory disturbances, (2) description of the pulsation of the liver, (3) distinguishing between the different varieties of gallop rhythm, (4) explanation of the mechanism of the apex beat, and (5) recognition of the "bruit de tabourka,"[1] the second heart sound, which occurs in the presence of syphilitic aortitis.

Potain long had been interested in the measurement of arterial pressure, which Karl Vierordt (1818-1884) had advocated and for which Étienne Jules Marey (1830-1904) had provided the means of evaluation. Marey's instrument, however, was neither precise nor practical. According to Vaquez, Potain learned "with joy" of Von Basch's invention of sphygmomanometry. Von Basch's instrument was a cumbersome affair, however, and Potain improved upon it and made its use practicable.

Potain's development of the portable sphygmomanometer led him to new discoveries. It permitted him to demonstrate the reality of hypertension in Bright's disease, as suspected by Traube. It also enabled the detection of hypertension in other pathologic conditions to be made. Potain, moreover, demonstrated that hypertension and not the impaired function of the kidney was responsible for the almost constant cardiac hypertrophy found in renal sclerosis.

The mechanical genius of Potain is also shown in many other ways. He perfected an apparatus to count red blood cells (Malassez hematimeter). In studying the congestive form of pulmonary diseases, his intern Georges Dieulafoy had chanced on the discovery of pleural aspiration. Potain helped him perfect an aspirator with a vacuum apparatus (Potain's apparatus), which is still in use. By means of this new apparatus he was destined to add further to his renown. For a patient who had extreme dyspnea, Potain performed thoracentesis and completely withdrew the fluid by means of his vacuum apparatus. With another apparatus, which he had constructed for the occasion, he replaced the fluid by air progressively introduced to avoid undue expansion of the lung. He used the same procedure for twenty patients, obtaining equally favorable results. He can, therefore, be said to have perfected, and he later reported the first indisputable facts extant on the favorable influence of collapse therapy in the evolution of the treatment of tuberculosis.

Potain was a discreet writer and he worked for a long time before he announced his discoveries, feeling that he might be premature in his judgment. When he did express his thoughts in writing, however, they were generally in the form of short memoirs, and were models of exposition in clearness and style. One of these short memoirs, "Théorie du Bruit de Galop" (1885), it is our privilege to reproduce in translation.[2] We are also presenting to our readers a translation of his classic description of the bruits of the jugular veins, one of his earliest works, published in 1867.

[1] Gibson, G. A. *Diseases of the Heart and Aorta*, New York, 1898, The Macmillan Company, p. *27.
[2] *loc. cit.*, p. 632

ON THE MOVEMENTS AND SOUNDS THAT TAKE PLACE IN THE JUGULAR VEINS*

By

DOCTOR POTAIN

Associate Professor of the Faculty of Medicine, Physician to Necker Hospital

Gentlemen:

You have heard in our recent meetings two very important papers concerning the murmurs of vessels. The one of M. Parrot and the other of M. Peter. Both of these gentlemen have questioned the diagnostic value which is generally granted to vascular sounds in the diagnostics of anemia, and both are of the opinion that it is necessary to seek the immediate cause of these phenomena, whatever additional may be the share of the state of the blood itself. Each stated further that he believed he was able to specify this cause; M. Parrot maintained that it is due to insufficiency of the valves of the jugular vein; M. Peter, to a spasm of the venous or arterial wall.

I also desire to say a word on this subject, not so much to dispute the opinions of my two colleagues as to present, in turn, the results of researches concerning this question which I have carried out over a period of several years. Until now I have not considered my study far enough developed to present to you, and now I do not believe by any means that I have obtained a definite solution. But since the question of vascular sounds has been raised among us, I do not wish to withhold them any longer.

The results at which I have arrived are in agreement in some respects with the ideas presented by my colleagues; in others they do not agree. Like these gentlemen, I believe that vascular murmurs are not pathognomonic of anemia. As with them it has appeared to me that this murmur could exist and be found most intense in persons not at all anemic: whereas it is entirely absent in persons who are obviously anemic to a high degree. Exceptions were known and pointed out by M. Bouillaud in his first studies; but they are more numerous, without doubt, than is generally believed. I admit with my colleagues, following the opinion of M. Chauveau, that the veins are the site, not only of continuous murmurs but also of the majority of intermittent murmurs which are heard over the neck.

I am in complete disagreement with each of them as to the further specification of the mechanism of the venous murmurs and as to their clinical value. Because, on the one hand, my observations do not permit

me to admit that the ordinary cause of these murmurs is concerned with a venous reflux, nor with a spasm of the great vessels; on the other hand they lead me to believe that the variable proportion of blood corpuscles cannot but have a direct and markedly appreciable influence on the production of these murmurs; and, furthermore, that certain types of these abnormal sounds may moreover be considered as characteristics of anemia.

For the remainder, the points of this question which our colleagues have taken up are concerned at the same time with the mechanism and with the clinical value of the vascular murmurs; we shall return then to these two aspects of the problem. Let us see at first that which concerns the mechanism.

I OF THE MECHANISM OF VENOUS VASCULAR MURMURS WHICH ARE HEARD IN THE NECK

When one observes carefully the portion of the supraclavicular region which the jugular veins and the carotid artery cross, one may frequently note three distinct phenomena through which are revealed the motion by which the blood is quickened in the vessels: (1) visible oscillations; (2) a thrill sensible to the finger; (3) normal and abnormal sounds revealed by auscultation. Since an analysis of the movements and the peculiarities which the purring fremitus shows may throw some light on the mechanism of the murmurs, I have applied myself to study them with care in a large number of individuals, and here is what I have found concerning this subject in repeated observations.

(1) The visible oscillations in this region consist of a series of filling and collapses, sometimes prominent and easy to recognize. In some persons they are appreciable only in the lowest part, that is, nearest the clavicle; in others they are seen distinctly over a large portion of the vessels; exceptionally, one may follow them over the course of the external jugular.

Because of their unequal amplitude, they are not always equally obvious and sometimes one or more are completely lacking; but when they are well marked, when no affection of the heart alters the rhythm and when the pulse is not too fast, their study is generally quite easy. There is found then, aside from the slow oscillations caused by the respiratory movements and simultaneous with them, the following sequence of movements which is repeated with constant and perfect regularity: at first a slow elevation, then two quick elevations, finally two deep depressions, after which the series begins again. Now each series of this kind corresponds to a cardiac cycle.

These impulses sometimes have such force and amplitude that at first it might be believed that they represent pulsations of the carotid artery or of the subclavian. But after a little attention one is soon convinced that they actually take place in the internal jugular. This is proved thus:

In the first place they are of a vague and diffuse nature, totally opposite to the idea of an arterial pulsation, and yet apparently, however extensive they may be, they are felt with difficulty by the finger which perceives with intensity pulsations of very slight extent either at the same level and nearby, or a little higher along the course of the carotid; in the second place, their rhythm does not resemble in any respect that of the pulsations of the carotid, the proof of which you shall see now in the totally different form of the tracings shown by the sphygmograph; in the third place and finally, a light pressure, suitably applied to the lower portion of the neck can impede them or suppress them entirely, while the pulsations of the carotid persist with all of their intensity.

These movements verified and their site established, it remains to seek an interpretation. It appeared to me that the most certain means would be to determine exactly their relationships with the different portions of the cardiac cycle. I studied them then in this relationship by combining palpation or auscultation of the precordial region with inspection of the cervical pulsations, and, in this manner, I arrived at these results. The first of the two sudden elevations immediately precedes ventricular systole, while the second coincides nearly exactly with it; the first depression takes place during the short silence, and the second, immediately after the second heart sound, that is, at the time of ventricular diastole; finally, the slow elevation which initiates the series, occurs in the middle of the long silence, that is, during cardiac rest.

This method of study permitted the clear establishment of the relation between the jugular pulsations and the movements of the heart, and I have found: that the two elevations observed correspond to the successive contractions of the auricle and ventricle, and the two depressions to the diastoles of these cavities. In fact, there can be little question but that the second quick elevation appears to follow the systolic impact and first heart sound immediately, and to coincide exactly with the pulse of the large arteries. As to the first, which precedes it, occurring before the contraction of the ventricle, it certainly cannot have for a cause an action which it precedes, it must then be attributed to the systole of the auricle. Finally, the two depressions appeared to me to occur at the precise moments which we know are occupied by ventricular and auricular diastole; and their appearance, at this moment, is explained without difficulty, because it is entirely natural to see the veins collapse suddenly at the moment when the blood that they contain is propelled toward the cardiac cavities, which pass suddenly from a state of contraction to one of relaxation.

But the comparative study of venous pulsations and movements of the heart, even though these movements are of slow frequency, is a very delicate thing, because it requires the comparison of impressions received by two different senses, which is always difficult. Moreover, I

have become accustomed by patient observation to understand clearly the sequence and relationships of these somewhat complicated movements, but I could not expect to demonstrate them readily. Furthermore as soon as the heart beat is accelerated a little, this study becomes impossible and the relationships are appreciable with great difficulty. I decided then, in order to study them with more precision and in a more demonstrable manner, to use the valuable sphygmographic apparatus which we owe to our colleague, Marey.

The idea of obtaining a graphic representation of the jugular pulsation is not at all new. In Germany, Bamberger, Geigel, and Friedreich have already employed it in the study of exaggerated pulsations seen in certain cardiac diseases, and Friedreich has shown that one may obtain a similar tracing in the absence of a pathologic venous pulse. But these observers were content to apply the sphygmograph designed by Marey for recording the radial pulse to the region occupied by that vessel. There resulted, in the first place, many difficulties in the application of the instrument; in the second place, a complete impossibility of determining then with any certainty the significance of the various portions of the tracing and their relationship to the different motions of the heart. But, this last point being exactly that which I sought to establish, I had to approach differently.

I employed the addition which Marey has made to his instrument to transform it into a cardiograph. I recorded the jugular pulsations with the help of a small glass funnel which acted as a stethoscope and transmitted the impulses received to the tambour of the instrument through an India-rubber tube. Simultaneously I applied the same sphygmograph to the radial artery to which were transmitted the pulsations of the jugular. Then I arranged things in such a manner that the two levers, that of the jugular and that of the radial, would write their tracings at the same time, on the same paper, and one above the other. Not content with this, I placed the funnel, or another instrument better arranged, over the precordium and thus recorded the impulses of the heart simultaneously with those of the radial pulse. Finally, for more certainty, while the pulsations of the heart were being recorded, I placed the sphygmograph directly over the carotid in cases in which this artery was easily accessible. It only remained to compare these different tracings and to superimpose them with care by means of a very precise method, but which would be too long to discuss here, to see coincidences established in some manner of themselves and in the most strict fashion which can be imagined.

Here, Gentlemen, is an example which has been enlarged[1] to show to you:

You may see at once in the first tracing of this figure (I) the exact reproduction of the movements which I just now described to you after

[1]The figure placed here is a reproduction of the tracing in its original size.

simple inspection: a progressive rise (A); two short elevations of little extent (B) (C); then two deep depressions (D) (E). You will note further that the same movements are reproduced at each cardiac cycle, modified only by the respiratory oscillations which are added and which cause some changes in the line as a whole without ever suppressing any of the details mentioned. In the same figure are reproduced, with the help of the procedures which I have outlined to you and with the coincidences marked exactly by vertical lines, the radial pulse (II), the carotid pulse (III), the impulse of the apex of the heart (IV). I believe that on comparing these four tracings one may determine the precise significance of each part of the first, as you may judge.
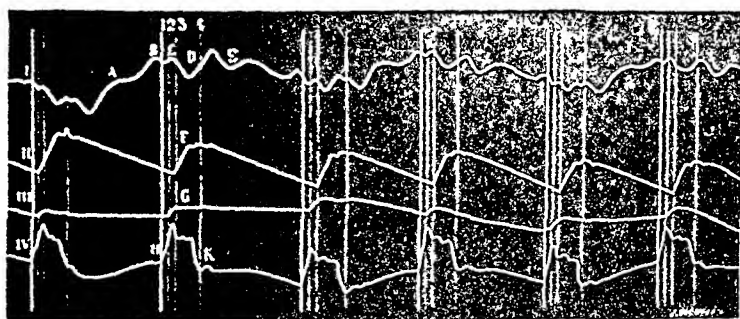
Fig. 1.

I. Pulsations of the jugular vein.
II. Pulsations of the radial pulse.
III. Pulsations of the carotid.
IV. Pulsations of the apex of the heart.

A. Progressive repletion of the vein.
B. Elevation caused by *contraction of the auricle*.
C. Elevation caused by *contraction of the ventricle*.
D. Depression produced by *diastole of the auricle*.
E. Depression produced by *diastole of the ventricle*.
H. Beginning of ventricular contraction.
K. End of ventricular contraction, occlusion of the semilunar valves, beginning of diastole.

1. Line of beginning of ventricular systole.
2. Line of the carotid pulse.
3. Line of the radial pulse.
4. Line of *ventricular* diastole.

To show at first that the tracing (I) represents the pulsations of the jugular and not those of the carotid, it will suffice, in the absence of other proof, to compare it with that obtained when the instrument is placed over the carotid itself (III), and to note that the latter does not show any details which the former shows, and does not resemble it in any manner; further, to observe that the movement (B) takes place at a time when no impulse has been produced in the arterial system, because the ventricle (H) has not yet begun to contract.

If we now analyse the jugular tracing, we shall see there clearly what follows: (1) the first quick elevation (B) precedes the radial pulse considerably (F); it precedes the carotid pulse (G) also, although by somewhat less and most remarkable yet, the beginning of ventricular systole (H); consequently it could not be brought about by this systole which it precedes. On the contrary it is produced at the precise instant where physiology tells us that the contraction of the auricle takes place, and since there is no motion of the ventricle at this moment, it must be attributed necessarily to the contraction of the auricle. It is thus readily explained by the light reflux which auricular systole causes in veins near the thorax; (2) the prominence (C) immediately follows ventricular systole (H), and coincides exactly with the carotid pulse (G). Like the latter, it is the result then of ventricular systole, perhaps the systolic movement is transmitted directly to the venous system at the moment when closure of the tricuspid valve takes place, perhaps it is transmitted indirectly by the compression which the arterial trunks in diastole certainly exert on the venous trunks which they are near, perhaps, finally, it results from these two modes of action combined; (3) the first depression (D) takes place in the interval between the vertical lines 3 and 4 which indicate, the one, the beginning of the radial pulse (F), the other, the moment of ventricular diastole (K); that is, it is produced during the time when the ventricle contracts (HK), and in the moment when the carotid artery is in complete diastole (G). Consequently, it cannot have its cause either in the contraction of the artery or in the relaxation of the ventricle. But it corresponds precisely to the time when diastole of the auricle takes place; it should then result from the rapid flow of venous blood into this relaxed cavity, an afflux which immediately empties the veins near the thorax; (4) finally, the second depression (E) occurs a little after ventricular diastole (K), and it cannot be very well explained except by the new impulse which this diastole brings to the blood contained in the auricle and which is transmitted to the veins of the neck.

The tracing which we have analysed was taken on a woman in childbed in l'Hôpital Necker, in whom the pulse, very much slowed, beat only 40 times each minute. I have chosen this case to present to you first because this extreme slowness is especially helpful in the study of the coincidences which I wished to establish. Those which I have obtained in other cases of the same kind, are entirely identical; but, since you may think that the special state of the circulation in women in childbed might give rise to pulsations in the vessels which would not exist in the absence of this state, here on a second plate (Fig. 2) is a tracing taken from a man in whom the pulse, slowed by icterus, beat 46 times per minute.

You may see at once that the form of the tracing is entirely similar to that of the preceding one, that is, the elevations and depressions take

place in the same manner and in the same order; you note, further, that the coincidences are the same. Indeed, you see, as in the preceding one, at A the slow rise which corresponds to the absolute rest of the heart and which indicates the slow filling of the vein; at B the sudden elevation which precedes ventricular systole (II) and must be attributed to the contraction of the auricle; at C, the elevation coincides exactly with the systole of the ventricle; at D, the first depression that takes place during this systole and which represents the diastole of the auricle; finally, at E, the second depression which corresponds exactly with ventricular dilatation (K).
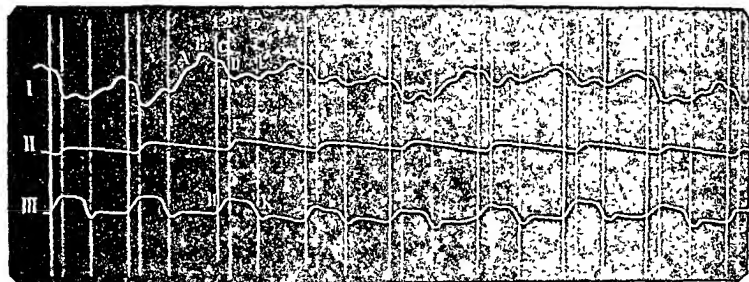
Fig. 2.

I.   Jugular vein.
II. Radial pulse.
III. Apex of the heart.

A. Progressive repletion of the vein.
B. Contraction of the auricle.
C. Contraction of the ventricle.
D. Dilatation of the auricle.
E. Dilatation of the ventricle.

1. Line of ventricular systole.
2. Line of the radial pulse.
3. Line of ventricular diastole.
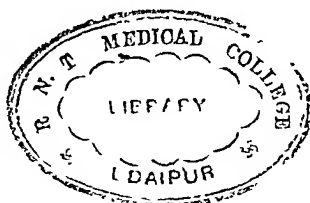
Finally, it is not necessary to have a pathologic state to show the pulsations of the jugular vein with this completeness and with all of the details which I have shown you in the preceding tracings. I show you, in proof of this, one which I have taken on myself (Fig. 3).

The elements of the curve are a little closer together, because my pulse is more rapid than that of the patients on whom I made the preceding tracings: but they have the same form, they exhibit the same order and are easily recognizable. Thus one may note at A, the slow elevation produced by the progressive filling of the vein; at B, the quick elevation due to the systole of the auricle; at C, that of ventricular systole; at D, the depression of auricular aspiration; at E, that of ventricular aspiration; that is to say precisely all that which we have discovered in analysing tracings shown by the jugulars of women in childbed or of icteric persons with slow pulse.

not suffice here; I wanted a more direct proof, and here is the very simple artifice which gave it to me.

I have placed, as you see here, a small funnel of glass with an opening about the size of the bell of a stethoscope at one end of an India-rubber tube, the other end of which was introduced into the ear. I applied this flexible and transparent kind of stethoscope to the region of the vessels of the neck; and in this manner I could hear the sounds distinctly, while I had the eye fixed on the region, in fact on the same point over which I listened. Then, to make the movements of this region more perceptible, I fixed a little piece of colored paper, bent at a right angle, with a little water to the skin covered by the transparent bell of my stethoscope. This paper, having the function of a mobile arm, or if you wish, of a sphygmoscope, considerably augmented the apparent amplitude of the movements and made them easy to observe.

In observing in this manner, I have always seen and have always proven for the students, as well as for everyone else who has wished to lend himself, that *the murmur takes place at the precise moment when the skin sinks within the area of the stethoscope;* that it is double when there is a double depression and that, on the other hand, it ceases at the instant when the region rises. Is it not a proof at once demonstrative and quite simple, that the murmur is not produced at the moment of diastole of the artery and that it is not arterial, further that it cannot be attributed to a venous reflux, but, on the contrary, that it takes place at the same instant in which the vein is twice under the influence of aspiration by the cardiac cavities?

Further, if in auscultation of the vessels of the neck one listens to the heart sounds, which are frequently transmitted distinctly into this region, one may determine without difficulty that the intermittent venous murmur of which we speak here is always produced, not with the first sound but with the second; that is to say that it corresponds, not to auricular and ventricular systole, but to the diastole of these cavities; in such a way that, when it is double, one precedes the second sound and the other follows it immediately. Now, the reinforcements of the continuous sound behave in exactly the same manner.

On the contrary, arterial sounds which are also heard in this region are characterized by the fact that they coincide exactly with the diastole of the artery and they follow the first heart sound immediately. They are distinguished also from the others by a different quality of sound, by a special quickness in their onset, and by the very different influence which compression or the efforts of inspiration and expiration have on them, finally by the impossibility of transforming them into continuous murmurs.

Thus it comes about that one may distinguish three types of murmurs in the neck: continuous ones, which arise in the veins as many proofs show; intermittent venous ones, *single or double*, which correspond to the second portion of the cardiac cycle and coincide with the depressions in the

cervical region, that is to say with diastole of the auricles and of the ventricle; intermittent arterial sounds which are produced in the first portion and accompany exactly the impulse of the artery.

You see, Gentlemen, when one studies with care the murmur which is of particular interest here, that is to say the venous murmur of the jugulars, one finds, in its intermittences or in its reinforcements, only one possible interpretation: that each gust of the murmur or each reinforcement results from an acceleration of the venous flow brought about by diastole of the auricle or of the ventricle.

The murmur, as everyone knows, passes easily from intermittence to continuity under the influence of various causes; for example, slight changes in the degree of pressure exerted over the vessels. And this is readily understood. In fact by slightly increasing the pressure of the stethoscope, the blood, retained and accumulated in the vein above the obstacle, soon acquires a tension so strong that it then passes by this obstacle with a continuous movement, without moreover being influenced (by the lower pressure) in any appreciable manner, without the variations which the lower pressure goes through which is present below.

Nevertheless, independent of external modifications of pressure, the murmur is, in the same individual or in different individuals, sometimes continuous, sometimes single intermittent, sometimes double intermittent, or continuous with reinforcement; and it is surprising that a phenomenon, the rhythm of which is regulated by causes working at all times and everywhere, should nevertheless be so variable. The auricle and ventricle always contract and dilate; why does not the murmur always have a double intermittent rhythm, which is the consequence of these movements? Why? Because the aspirations and refluxes or retardations which originate in these cavities are not the only influences which the circulation of blood in the veins obeys; because there are others which operate in a more or less continuous manner, such as: the thoracic aspiration which is exerted on the large venous trunks contained in the chest, the state of the capillary circulation, the tension of the soft parts which surround the vein, the pressure which they exert, certain arrangements of the vein itself. And, according to whether some or others predominate, it is conceivable why there should be a tendency to intermittence or continuity. But these influences are not always easy to determine when one comes to particular cases. Thus, not possessing precise data on this point, I shall not speak further. I am satisfied, for the present, having shown you, to rest on the proof which I believe positive, of the cause to which should be attributed the frequently intermittent rhythm of murmurs heard over the jugular veins.

After all, this method of interpreting intermittent murmurs of the neck is not exactly a novelty. In his paper of 1858, M. Chaveau already pointed out the influence which ventricular aspiration exerts on the flow of blood in the jugulars. But this able investigator doubted that diastole of the auricle

also had any action of this type. Moreover his demonstrations did not appear sufficient beyond doubt, since they seem not to have carried personal conviction. This is the reason that I believed it necessary to return again to this subject and that I wished to study it anew.

The facts which I have presented to you are not, as you know, Gentlemen, in accordance with the theory which has been presented to you by M. Parrot. My colleague and friend believes, as you know, that the intermittent murmur or the reinforcements of the continuous murmur are the results of a reflux brought about in the jugular veins either by the contractions of the auricle, or by those of the ventricles, when there is insufficiency of the tricuspid valve. As to the continuous form, it results, according to him, from a succession of refluxes, and of affluxes exaggerated by the preceding refluxes.

Suitably to apply to observed facts, this new interpretation is faulty: first, in those cases in which it assumes an insufficiency of the venous valve, the murmur, supposedly produced by the contraction of the auricle, should immediately precede ventricular systole and, in those in which it further concedes tricuspid insufficiency, it should coincide exactly with this systole; secondly, it is also faulty, where the rhythm is double and intermittent, in that the first of the two murmurs should coincide precisely with an elevation of the region to which the stethoscope is applied and the second with a depression. But this never takes place. On the contrary, I have always seen, that when there are two sounds, the first follows the first valvular sound after an interval, and the second follows the second heart sound immediately; when, on the contrary, the sound is single, I have always heard it begin either immediately before or immediately after the second heart sound, never after, immediately before or exactly with the first. Finally, when the circulation was slow enough to permit a good analysis of these phenomena, I have always seen the single or double sound accompany the single or double depression of the region.

To the thesis which I adopt, M. Parrot opposes the difficulty of conceiving that a sufficiently continuous movement of blood could exist in the jugular vein to give the perfect continuity to the murmur which is sometimes found; he believes that each systole of the auricle or of the ventricle should necessarily bring about a perceptible interruption in the flow.

That would surely be inevitable if the jugular opened directly into the auricle; but this is not the case. Between this vein and the heart are interposed the great venous trunks inclosed within the thorax and constantly subject to thoracic aspiration; now these venous trunks, having the function of reservoirs and of a source of constant supply, in certain circumstances may weaken or cause the disappearance of the effect of the reflux which results from cardiac contractions. Moreover, as M. Monneret formerly observed, it should be even more difficult to conceive

of a perfectly continuous murmur produced by a succession of flux and reflux. Whatever might be the rapidity with which these alternating movements might succeed one another, there must always be a dead point, as is said in mechanics, that is to say a moment of repose, and no matter how short you wish to assume, the ear will always be able to grasp it. I have tried in inert tubes and in the jugulars of cadavers artificially to produce a continuous sound by the most rapid possible succession of two currents in opposite directions; never have I succeeded. Further, it has been demonstrated experimentally, as you may now see for yourselves, that a murmur differs in quality and intensity according to whether one listens at a point above or below the point at which the murmur originates. One should always observe in a murmur produced in this fashion an alternate change of quality and intensity. That is to say that truly uniform continuous murmurs absolutely resist the method of interpretation proposed by our colleague. Further, inspiration and expiration which influence the intensity of murmurs in such an obvious manner by modifying the blood flow, according to this hypothesis should influence unequally the two parts of the continuous murmur. Expiration should reinforce the murmur of reflux and weaken that of afflux toward the chest, while inspiration should act in an opposite manner; but this is not the case. Inspiration reinforces the murmur in a uniform manner; expiration weakens it in the same manner.

Thus, then, in addition to the fact that the theory of my colleague is in formal opposition to facts which I believe are well established, it is of itself open to serious objections and it is for that that it is not possible for me to accept it.

The theory of arterial or venous spasm which M. Peter is concerned with reviving, undoubtedly is not in as evident opposition to the previously mentioned facts; nevertheless, in its turn it raises objections no less serious. You have seen, indeed, that it rests chiefly (on the following): first, that without it one may not explain how the vascular murmurs show variations so frequent, so sudden, so completely independent of changes which might take place in the composition of the blood; second, on the contractility of vascular walls which is generally admitted today.

It is incontestable that one cannot establish any precise and exact relationship between these murmurs and the state of the blood, and that these abnormal sounds frequently show an extraordinary variability; on the other hand, venous contractility is undoubtedly a fact completely demonstrated in physiology. But you shall see, Gentlemen, in what remains for me to say, that these apparent anomalies may have explanations entirely different from that which has been given by Laënnec, and, in my opinion, better founded.

Moreover, since nothing can be shown directly as to whether or not the venous wall is in a particular state of contraction in individuals in whom

the murmur is found in the neck, this contraction always remains in the state of a pure hypothesis. Consider further that this hypothesis is not successful and that it is very poorly applicable to facts which it is supposed to explain.

In fact let us admit that a contraction may take place in the jugular vein of a subject being examined; this contraction either extends the full length of the vessel or it is limited to a circumscribed portion. In the first case, it is probable that it would make the murmur disappear, if one existed previously; because its effect would be to retard the flow of blood and to diminish the quantity which would pass through the vessel. In the second, after all, the pressure would not do anything more than the pressure of the stethoscope can do and to a degree which the stethoscope can always attain since the pressure of the stethoscope can always be carried to the point of absolute suppression of the murmur by effacing the lumen of the vessel. If, then, venous contraction is the only cause of murmurs; since the pressure of the instrument can do the same as venous contraction, the stethoscope should be able to produce a murmur in every case and there would be no question as to why certain people do not show it at all although they may be quite anemic.

This theory of vascular spasm is, then, a hypothesis without possible demonstration, which is entirely unnecessary to explain the facts and which gives a poor explanation when one attempts to apply it to them; thus I believe that it should be absolutely rejected.

II. OF THE CLINICAL VALUE OF VASCULAR MURMURS

In what concerns the clinical value of vascular murmurs, that is to say, their relationship to anemia, I agree with the opinion of my two colleagues, without, however, adopting it entirely. M. Parrot distinguishes two cases: in one, the murmur has absolutely nothing to do with the state of the blood; its cause is insufficiency of the valves of the jugular vein, a purely accidental insufficiency. In the other it is not without some relationship with anemia; but this is a distant and indirect relationship, since it results from insufficiency of the tricuspid valve, an insufficiency which should be rather frequent in persons affected by anemia. As to M. Peter, the relation of the murmur to anemia is always indirect, distant and therefore contingent. The immediate cause of the murmur is a spasm of the vein which the poverty of the blood promotes, as it promotes all spasmodic states.—As for me, I continue to believe that in the hydremic state there is a condition much more directly favorable to the production of murmurs, and that this state, carried to a certain degree, can produce murmurs without any vital act, without any modification of the caliber of the vessels. Nevertheless it should be added that conditions wholly apart from the state of the blood, and further, quite complex, contribute, each for its part, to favoring or impeding the production of vascular

murmurs; so that the appearance of murmurs is the result of multiple circumstances which cannot be revealed except by a careful analysis.

Thus the inconstancy of the murmur in conditions apparently identical at least as to the state of the blood. Also the impossibility of finding a cause which is single and always the same for this phenomenon.

Gentlemen, permit me to begin this subject with certain developments. This seems necessary to me because directly opposite opinions are found here sustained by considerable authority. We must then seek and discover the reasons for these divergences which are surprising when it is borne in mind that it is only a question of establishing a plainly physical fact and of understanding what relations it may have to certain conditions of the blood.

I do not wish to repeat the history of this question here. Since the discovery by M. Bouillaud, who first reported it,* with wise restrictions, of the relationship habitually existing between intense murmurs and anemia, everyone knows that the most divergent views on this subject have arisen among physicians.

Some, such as Gorup-Besanez, Skoda, M. Chauveau, and recently in your presence, M. Peter, reject vascular murmurs absolutely from the symptomatology of anemic states; others, on the contrary, maintain with MM. Barth and Roger that, "continuous murmurs are the most certain indication of advanced chlorosis and of anemia with diminution of blood corpuscles."

This question deserves examination because it arises constantly at the bedside and the solution may, in many cases, considerably influence our practice. But, in order to procure the necessary exactitude, it is necessary in anemia to distinguish two different states, the influence of which on the production of murmurs is certainly very different, thus: true anemia or the diminution of the volume of blood, and hydremia or the diminution of corpuscles.

A. *The Relation of Murmurs to True Anemia.*—In spite of the contrary statements of M. Vernois, M. Chauveau, and M. See, I do not believe that true anemia can give rise to the vascular sound which we are considering here; that is, continuous or intermittent venous murmurs. It is known that it is not at all possible to confirm the isolated existence of this form of anemia, except when a large hemorrhage takes place in a person with good health. Now I have practiced auscultation a large number of times in these conditions and I have never seen the murmurs of which we speak appear at that time when they had not been present previously. On the contrary, I have seen, as M. Bouillaud pointed out in his first work on vascular murmurs, the murmur disappear under the influence of a blood loss of some importance, to return much later, during the period of restoration. This morning on the obstetrical service of the Necker

Hospital, I saw a woman who had an abundant hemorrhage and who did not show any trace of a murmur over the jugulars. Besides, do we not see every day that in those of our patients in whom this type of anemia predominates especially, the phthisical, the cachectic of any type, they are precisely those in whom the jugular murmurs show the least intensity? This first point appears proved to me; moreover, it is not in dispute. Let us pass then to hydremia.

B. *The Relation of Venous Vascular Murmurs to Hydremia.*—That which we now know of the relation of vascular murmurs to hydremia rests principally, nearly exclusively, on the researches of M. Bouillaud and M. Andral. The analyses of Gorup-Besanez and certain ones which MM. Becquerel and Rodier have made on the blood of chlorotics have been opposed to them already. And now M. Parrot and M. Peter present their clinical observations to you as absolutely contradictory to the opinion of the masters whom I cite. But there should be no contradiction in well observed facts; the contradiction can be only in the interpretation which is given them and in the deductions made. Let us see then if we may discover them.

M. Bouillaud immersed the hydrometer of Baumé in the blood of twenty-seven persons who had been bled for various febrile affections, several successive bleedings. In each individual, the murmur appeared and was increased according to the repetition of bleeding and the further depression of readings of the instrument. Further, each time that the hydrometer showed more than 6¼ the vessels had no murmur; on the contrary, every time that the hydrometer fell below 5½, the murmur was present. Between these two limits there was an equal number of subjects having the murmur and of others who did not show it. But if these two groups of data are analysed further and one takes the reading of 5¾ for a median point, it is found that among those patients who showed a specific gravity of the blood above this figure, only one had a murmur and eight did not have it, while on the other hand, those in whom the density of the blood was less, seven had a murmur and only one did not. From this one may rigorously conclude that, under the conditions in which M Bouillaud worked, that is to say in the patients who were bled, the murmur became better manifest as the blood was impoverished further by bleeding; that this sound appeared only exceptionally when the density of the blood taken with the hydrometer was above 5¾ and that it *was almost constant when it descended below* this figure. By innumerable observations M. Bouillaud has discovered that intense murmurs are almost always found in individuals, men or women, who show the signs of chloranemia to a high degree. And as it is known, moreover, that the blood of chloranemia lacks especially red corpuscles; that it is the lowered proportion of the same corpuscles in the blood of persons subjected to bleeding that chiefly depresses the density

of that liquid; it results that chloranemics and patients bled in any large quantity are, in this respect, in the same condition; that they have blood equally poor in corpuscles and that this special poverty is accompanied equally in both by vascular murmurs, there should be in all cases a strict relationship between the presence of murmurs and this state of the blood.

But M. Bouillaud has not said in any manner and he has never contended that the murmur is an absolutely constant phenomenon in every degree and every condition of anemia, nor that it is never met with in persons exempt from this pathologic state, still less did he say that the intensity of the murmur should be strictly proportional to the degree of hydremia. Because, to use the master's own words, it is only when this phenomenon is present "in a high degree that one may affirm the existence of a chlorotic or anemic state, and reciprocally."

Likewise, the analyses of M. Andral have shown that in general the murmur is constant when the number of corpuscles falls below 80 p. 1000; that the murmur is likewise more frequent as the proportion of corpuscles, in falling, more nearly approaches this figure; that finally the continuous murmur is found more frequently in relation to the intermittent murmur as the more pronounced states of hydremia are observed. But this does not imply that murmurs always accompany anemia, that they do not exist without it, nor that they give an exact measure of it by their intensity.

There is nothing contradictory between these clinical rules and the observations which have been presented to you by M. Parrot and M. Peter. The observations of these gentlemen have shown us a certain number of persons without a murmur with the external characteristics of anemia or subjects who had intense murmurs with the appearance of excellent health. Now these facts, in some respects, being implicitly compromised by the rules in this regard which M. Bouillaud and M. Andral have established; since, according to them, the moderate degrees of anemia, those in which the figure is above 80 p. 1000, do not have the vascular murmur for a constant symptom. We accept them then, and we acquiesce so much the more willingly, for there is not one among us who has not, undoubtedly, been surprised more than once by facts of this nature. In collecting such a large number, our colleagues have given us a useful lesson for which we should be grateful to them. It will caution us against an error which we are, I believe, rather generally inclined to commit; that of easily granting, in practice, to this simple and easily determined sign, an absolute diagnostic value which it should not have, and of not taking enough trouble to determine its value by analysing the causes and conditions present. I have already made some efforts to combat this too common tendency in the article on anemia which I prepared for the *Dictionnaire encyclopédique des sciences médicales.*

Are we to say now that it is necessary to give up the view of relations established between the murmur and the hydremic state? Is it necessary to reject all of the positive results of experiments and analyses which I have just now cited? Is it necessary to seek the sole cause of abnormal sounds in circumstances independent of the state of the blood? For my part, I do not think so.

Only one serious objection can be made to the analyses of M. Bouillaud and M. Andral; it is that they deal with a number of cases relatively too restricted for the exceptions to the law which they formulate to be able to show themselves, and there does remain some incertitude as to the value of murmurs in the diagnosis of anemias of moderate intensity; since they had for subjects, patients affected by various conditions which further complicates the question. As to the contradictory analyses of Gorup-Besanez or of Becquerel, we may leave them aside because they deal with a number of cases even more restricted. To decide the question finally, it is necessary then to make a very large number of analyses, especially relative to murmurs of medium intensity, bearing comparatively on persons in the physiologic state and on patients simply anemic. For to estimate the state of the blood with some certitude merely according to rational symptoms, does not indeed require much thought. We know that the external appearance of the patient is one of the least dependable of the signs of anemia. M. Bouillaud and M. Andral have both sufficiently emphasized this fact. But in order that the analyses may be practicable it is necessary to have a procedure for measuring corpuscles which can be used with absolutely insignificant quantities of blood. But unfortunately we do not have such a one. The procedure for enumeration proposed by Vierordt is not at all practicable; the more simple ones of Welcker, de Nasse and de Panum require too much blood, and the criteria which I have established to make these methods of research applicable clinically have not been met up to the present, with the inability to obtain instruments constructed with enough precision. Montegazza in Italy, appears to have been more fortunate but he has not, to my knowledge, applied his instrument to this problem.

I have then attempted to approach this problem in an indirect manner. It is disputed whether or not the decrease of corpuscles in the blood is of any importance in the production of murmurs. I wished to find out if blood flowing through an inert tube would produce more or less of a murmur according to the quantity of corpuscles present. To that end I have constructed an apparatus which I have had brought before you and with which you may experiment in a moment. It is made essentially, as you see, of a vertical tube of thin India-rubber of about the caliber of the internal jugular of an adult. To this tube I have joined perpendicularly another of smaller caliber which, applied to it simply, does not communicate with its cavity. This serves as a stethoscope, the other end be-

ing applied to the ear, and permits one to hear the murmurs which take place in the first tube without exercising the least pressure on it, without displacing it the least. With a clamp of limited pressure, I can establish, at whatever point of the tube I wish, a narrowing of a perfectly determined degree and which I can vary at my will. Finally, this tube is connected with a glass reservoir of known capacity which I can fix at any height above the point of auscultation. To complete the apparatus two vessels, which may be put in communication with this common reservoir contain, one, serum of beef blood, the other, defibrinated blood, that is to say, serum with corpuscles. The serum and defibrinated blood were taken yesterday at the slaughter-house from the same animal. After having produced a certain narrowing with the clamp above the point of auscultation, at first I let pure serum run through the tube which immediately gives rise to a very intense murmur; then, while the liquid is still running, I substitute serum with corpuscles for the pure serum, and at the moment that the former comes into the tube taking the place of the latter, a considerable diminution of intensity of the murmur takes place. Finally, each time that these two liquids are alternated, the murmur is heard stronger or weaker, according to whether serum or defibrinated blood is flowing. One may, moreover, arrange things in such a way that the murmur produced by the serum is only of moderate intensity; then all sound disappears when the blood with corpuscles begins to flow. If one uses not pure serum but serum mixed with a small quantity of corpuscles, as is the case in anemias, one still obtains the same results, but with proportional differences of intensity.

Absolutely nothing changes here for the duration of the experiment except the proportion of corpuscles in the flowing liquid, and the murmur appears or disappears, increases or decreases constantly with each of these alternations. Does this not demonstrate, in a most rigorous fashion that blood which contains the fewest corpuscles is most apt to produce vascular murmurs?

Experiments of the same type, with inert tubes, have been made by various workers, notably Weber, M. Piorry and especially M. Monneret. But they have had the aim of explaining and not of demonstrating the aptitude of blood for producing murmurs; also the experimenters have used various liquids of different densities or viscosities, but not blood itself. Here the demonstration is direct and should not, I think, allow any doubt on this particular point of the question: that the quality of the blood, as to its greater or lesser content of corpuscles, *may* influence the production of murmurs, an influence absolutely independent of any vital act of contraction, dilatation, compression *etc.*

But why does the flow of blood richer in corpuscles produce fewer murmurs? Is it because it is less apt to vibrate, as is generally believed and as is accepted by M. Chauveau? Is it, as M. Monneret believes, be-

cause it flows less rapidly? In this regard, the same apparatus will give us a solution and a precise demonstration. You have been able to note, when serum and defibrinated blood passed alternately, that the latter flowed more slowly. If I now change the height of the reservoir without changing any of the other conditions of the experiment, I can likewise change the speed of flow. Now, when we have arranged these changes in height in such a manner that the rate of flow is the same for the two bloods, that is to say that the quantity of liquid contained in the reservoir flows out in the same number of seconds, you shall see that the intensity of the murmur does not change and that it apparently remains the same, whatever the quality of liquid employed. In this instance, only one thing appears to me to differ, that is the quality of the sound, the quality being more subdued, lower, in the blood richer in corpuscles.

Thus, in this apparatus, the poor blood makes more sound because it flows more rapidly; and, when the flow of the richer·blood is made equally rapid by increasing the pressure or suction which makes it move, the difference of intensity disappears, at least sensibly.

There is no reason to think that things happen otherwise in the blood vessels. On the contrary, there are reasons to believe that differences in velocity, due to the unequal proportion of corpuscles, are exaggerated considerably because of the multiplicity of obstacles which the capillaries present in place of the single obstacle which is in our apparatus. It must be admitted then that poor blood sounds more in the vessels, principally, and possibly solely because it flows more rapidly.

But you know, Gentlemen, that the velocity of blood flow in the living economy is not at all exclusively regulated by the quality of the flowing liquid. The force of cardiac impulsion, the width of the capillaries, the aid or resistance present in the chest are also conditions which strongly influence the course of blood in the vessels, and notably in the veins. We may add, that a local narrowing, followed by a relative dilatation, is necessary for the production of a murmur, as M. Chauveau has shown, the proof of which you may easily obtain in the apparatus before your eyes, that this narrowing, produced naturally or brought about by the instrument of auscultation, may vary by reason of a thousand circumstances; finally that a certain compression capable of slowing the flow of blood acting on the vessel either above or below the point of origin of the murmur, restrains or impedes its production. Thus you may judge, on the one hand, how different influences are added together and combined in order to unite to give rise to the phenomenon with which we are concerned, or to modify its manifestations; on the other hand, how the mode of action of these diverse influences probably amounts in all cases to causing variations in the speed of flow at the point of auscultation.

Thanks to these considerations, is it not now easy to understand how, within certain limits the presence of murmurs is connected with the existence of anemia, and how, nevertheless, this symptom cannot constitute a certain sign nor a measure of intensity.

One of the proofs of this latter fact, which for my part has impressed me very much, is that which is found quite frequently in the course of typhoid fever. Almost all persons attacked by this affection show a very marked murmur during the first period, that is to say the first septenary of their disease. I might almost say all, because I can scarcely recall any exceptions to this rule, after having done auscultation on a very large number of patients. It seemed difficult to believe that the murmur was here the expression of an anemia of any general type, or that typhoid fever would attack anemic persons almost exclusively. But this difficulty is no more; a greater one soon arises. In proportion as the disease progresses; in proportion as the turgescence of the face so remarkable in the first days disappears; in proportion as the patients become pale and lose their color, made anemic by the disease, by the diet and sometimes also by the method of treatment; also in proportion the murmur, far from being augmented as one should expect, on the contrary becomes weaker, is subdued, is diminished, and sometimes disappears completely. Now, if the murmur of the first days was a pure symptom of anemia, there is no way of understanding how it becomes weakened and ceases while the anemia obviously increases. We are obliged to admit that the sound heard at first did not arise from anemia, but rather from the state of the circulation; facile and rapid, in the first days, across the dilated and turgescent capillary system; on the contrary feeble and slow when the weakness of convalescence arrives.

Now that we may attempt to picture to ourselves the combination of conditions which regulate the production of venous murmurs, I believe that they may be reduced to two principles: (a) The rapidity of blood flow in the vessel where the murmur occurs; (b) The arrangement of the vessel itself and of the surrounding parts.

a. It is easy to find more than one convincing example of the increase in the venous murmur caused by acceleration of blood flow.—For if, on auscultation of the jugular, one exerts a sudden pressure over a certain extent of this vessel above the point of auscultation, the murmur is completely arrested. But, before disappearing it is reinforced, as a sort of a gush, because the pressure which forces out the blood contained in the vein makes it pass more rapidly beneath the stethoscope.—Another example. I have found in myself and in various patients that if, having placed the stethoscope over the inguinal region in such a manner as to hear the venous murmur (a murmur that can be heard wonderfully well in this region, although the contrary is generally maintained), if, I say, the muscles of the leg and thigh are made to contract forcibly at this moment,

there is heard, at the same instant, a very obvious continuous murmur, sometimes quite intense, which immediately fades away and disappears. This is because the muscles in their contraction, force out the blood contained in the veins of the inferior extremity and precipitate it toward the crural vein where the flow consequently undergoes a sudden acceleration. Now it cannot be a question here, as some have wished to believe, of a reflex caused by the phenomenon of effort. If this were so, the sound would appear equally on the two sides, whereas it never appears except at the root of the limb of which the muscles contract.—Finally if, after having let one of my arms hang for some time and become congested, the rest of the body being extended horizontally, I then raise it rapidly in the air, I feel a very pronounced thrill in the subclavian vein and I also hear an obvious murmur. Now the transitory thrill and murmur can result only from the acceleration of blood flow in the vein. Indeed, they cease after an instant and are not reproduced unless the experiment is repeated at intervals long enough for the blood to have time to accumulate again in the veins of the extremity in its lowered position.

As these special influences created by the experiment should be left aside when we come to auscultation of the neck practiced in view of diagnostic indications, we may assert that the rapidity of blood flow in the jugular veins depends especially on the three following conditions: (1) the force of cardiac contraction; (2) the greater or lesser facility of blood flow through the capillaries; (3) modifications of thoracic aspiration.

The first, the importance of which one cannot doubt, is quite difficult to appraise in practice; because we do not have any precise and positive method of measuring the energy of cardiac contractions. We can only cite examples, such as the disappearance of murmurs in the syncopal state. —The second is without question the most important of the three, that which exerts the greatest and most constant influence on the production and intensity of murmurs. It is that which is obviously at work when murmurs are intensified by the influence of high temperature, of certain fevers which impart a special activity to the peripheral circulation (typhoid fever, eruptive fevers, etc.) or even physiologic fever which follows muscular effort and works in the same way. We are concerned with it again where an intense cold weakens the venous murmurs and makes them disappear for the time being. Its mode of action is easy to understand, because, the greater the abundance of blood passing through the capillaries, the more it returns through the veins; so that the rapidity of venous blood flow is thus accurately regulated by the activity of the capillary circulation. Thus it is here, without doubt, that we must seek the cause of many of the changes which take place in murmurs, without apparent cause and without supposed modification of the composition of the blood; changes which have embarrassed M. Peter so much. It is here, I believe, that we must transfer the spasm imagined by Laënnec, of which our colleague has taken the de-

fense. Here, however, spasmodic contractions of the vascular walls suppress the murmur rather than produce it as Laënnec believed and as our colleague thinks with him.

With respect to this we must remember that the tonicity of the blood capillaries is not the only condition upon which the velocity of blood flow in these vessels depends. Further, we have shown experimentally, how the variable proportion of corpuscles may accelerate or retard the flow of blood in this part of the vascular system.

The influence of thoracic aspiration on the venous flow in the neighborhood of the thorax is a fact so well known and supported by so many proofs that it is useless to lay stress on it. As to the action on murmurs and its consequence one may easily establish in this, that inspiration frequently exaggerates them, perhaps gives rise to them, and that their increase is more marked as inspiration is made with more effort. The increase is such that quite frequently it is taken for a tracheal sound; and one can rid himself of this error in no better way than this, by pressing the vein above the stethoscope lightly, the vascular sound ceases completely, leaving only that which arises in the air passages. Expiration works in the opposite manner, and in proportion as it is done with greater effort. Also one never finds any murmur in asthmatic and emphysematous patients, in spite of the usually large volume of their jugular veins; one might say precisely on account of the distention which they show.

In short, every acceleration of blood in veins is favorable to the appearance of murmurs on auscultation; every slowing makes an obstacle; and the causes of acceleration or of slowing may be found: for one part in the energy of cardiac action; for another, in the activity of thoracic aspiration; in the third place, in the facility with which blood passes through the capillaries. Furthermore the latter depends on two things: first, the width and variable tonicity of these vessels; second, the degree of viscosity of the blood which results from the greater or lesser proportion of corpuscles present. But that is not all, and we shall see in the following section other additional influences arise.

b. Let us come now to influences which depend upon the state of the vein and of the surrounding parts. It is at first an incontestable fact, after the demonstration furnished by M. Chauveau, that the blood does not produce a sound in flowing through the vessels except when it encounters a narrowing, that is to say, a point over which it passes from a relatively narrow portion which it crosses rapidly to a much wider portion where it flows more slowly and under lower pressure. This condition is realized in the jugular vein at various points and in various ways; for example, at the inosculation of the vein with the brachiocephalic trunk; perhaps at the level of the insertion of the valves; or further, at points compressed by muscles or taut aponeuroses; or finally, and frequently, at the point of pressure of the stethoscope. The various positions as-

sumed by the neck certainly contribute to making the realization more or less complete. It should be added that, in the position recognized the most favorable for the production of murmurs, a slightly exaggerated rotation compressing the jugular of the opposite side, consequently tends to increase the activity of the circulation in the one left free and on which auscultation is done. It should be added that a very light contraction of the sterno-mastoid may, by compressing the vein, weaken or completely efface the murmur. Also as everyone knows, a slight displacement of the head is sufficient to cause this sound to appear or to cease suddenly. It is to this circumstance, frequently quite difficult to appreciate except with very close attention, that I believe it is necessary to attribute, in a large number of cases, the variability of abnormal sounds which has so much surprised observers, and which M. Peter believes cannot be explained without admitting a spasm of the large veins.—It goes without saying that the thickness of the soft parts which lie over the point of auscultation should also enter in some way into the intensity of the sound. So that the anatomical disposition of the parts exerts, as we see, a very complex influence on the manifestations of the sign which we have studied.

In the presence of so many different influences, near and remote, which unite with anemia in determining the appearance of vascular murmurs, or in modifying their intensity, one cannot expect to see this sign express faithfully the existence or the degree of alteration of the blood. To constitute a sign of anemia it is absolutely necessary, as Bouillaud has said, that the sounds should be "very intense." It is necessary, further, that one should be certain that no other circumstance independent of the state of the blood exist capable by itself of causing the appearance of the abnormal sound. Without analysing the conditions which give rise to it, this sign is of no value; in this regard I am in perfect agreement with my colleagues.

However, there exists a particular form of the murmur which I believe is quite characteristic of hydremia, it is the sibilant, musical murmur which M. Bouillaud calls "the song of the arteries." What makes me think thus is that this form has never appeared at all except in persons having elsewhere quite positive signs of anemia; this is the form in which it appears after loss of blood.

I have shown previously, in my inaugural thesis that when a subject has undergone any abundant hemorrhage, the murmur, absent at first, appears only after some time, it increases progressively during a certain number of days, then it decreases until it finally disappears. It increases in proportion as the blood, at first restoring its serum, becomes relatively poorer and poorer in corpuscles. It decreases when the corpuscles, in their turn reappear, returning the blood to its former composition. Now, during these periods of successive increase and decrease, one sees the

murmur successively take on the intermittent form, then the continuous, then the musical to return then to unmusical continuity, to intermittence and it disappears. The sibilant murmur, which is thus placed at the peak of this sort of curve, corresponds then precisely to the highest degree of alteration of the blood and consequently appears a little more significant than the other forms of abnormal vascular sound.

CONCLUSIONS

Before ending this communication already long, wherein I have not pretended to solve all of the questions which the problem of vascular murmurs brings up, allow me, Gentlemen, to summarize in some propositions that which the facts and reasons which I have had the honor to present to you, appear to me to lay down the most essential and the most positive:

1. Two kinds of murmurs are heard in the neck: arterial murmurs and venous murmurs;

2. Arterial murmurs are intermittent; venous murmurs may be continuous, intermittent, or continuous with reinforcements;

3. The reinforcements or repetitions of the venous murmur have no other cause than intermittent accelerations of blood flow in the vein;

4. These accelerations and their relation with reinforcement of the murmur are, moreover, demonstrated in a positive manner by the depression which appears, at the moment they take place, at the point of the region which corresponds to the vein. They result from successive aspirations set up in the venous system near the thorax by diastole of the auricle and ventricle, as the sphygmographic tracings show;

5. The murmur appears the more easily in veins in which the blood is poorer in corpuscles;

6. If the poor blood is more apt to produce murmurs, it appears to be nearly exclusively because it flows more easily and more rapidly;

7. The appearance of murmurs in the jugular does not depend solely on the poverty of the blood; it is subordinate further to other conditions, some of which are local, others general. All appear to act, directly or indirectly, so as to accelerate or retard the blood flow in the vein ausculted, or to produce the local narrowing necessary to cause vibration of the liquid;

8. One may not attribute any value to murmurs in the diagnosis of hydremia except when they show a rather greater intensity and when, moreover, one has taken into account circumstances independent of the state of the blood which might contribute to their production;

9. There is however a variety of vascular murmur the clinical value of which appears somewhat more positive and which seems to be connected more directly with hydremic alteration; it is the musical form of this murmur.
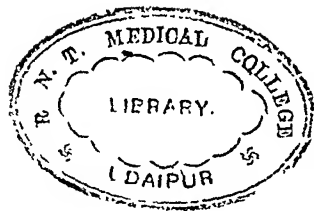
1867

T. LAUDER BRUNTON

THE INTRODUCTION OF AMYL NITRITE IN THE
TREATMENT OF ANGINA PECTORIS

SIR THOMAS LAUDER BRUNTON

(Courtesy St. Bartholomew's Hospital Reports.)

SIR THOMAS LAUDER BRUNTON

(1844-1916)

"He was a kindly Scot."

THOMAS LAUDER BRUNTON was born on March 4, 1844, at Bowden, Roxburghshire, Scotland. He was the youngest son of James Brunton, a gentleman farmer. Brunton was educated privately and in 1862 began his medical studies at the University of Edinburgh. He had a marked capacity for work and received many honors as an undergraduate. In 1866 he received the degrees of Bachelor of Medicine and Master of Surgery, and in 1867 he was granted the degree of Bachelor of Science. During the latter year he served as house physician under Prof. Hughes Bennett at the Royal Infirmary in Edinburgh. In 1868, after receiving the gold medal award for his thesis, "Digitalis with Some Observations on the Urine," he was granted his Doctorate in Medicine. In 1870 he obtained the degree of Doctor of Science.

From 1867 until 1869, besides doing some original research at Edinburgh, he spent part of the time in study at the leading medical centers of Austria, Germany and Holland under many able teachers including Ernst Wilhelm von Brücke, Isidor Rosenthal, Ludwig Traube, Willy Kühne, and Carl Ludwig. At Ludwig's new laboratory in Leipzig, Brunton derived the main inspiration of his scientific life. There he worked on the independent contraction of the arterioles and capillaries, making some experiments on the effect of amyl nitrite and sodium nitrite.

Brunton meanwhile experimented in his laboratory at Edinburgh on the influence of digitalis on blood pressure in animals. His instrument for recording this pressure was a simple mercury column. He also experimented on the effects of digitalis on his own blood pressure with the aid of the sphygmograph. Brunton's facile use of this instrument led him to discover the elevation in blood pressure which accompanies angina pectoris. By correlating the pathologic data thus acquired and by utilizing his knowledge of the pharmacologic action of amyl nitrite, he succeeded in discovering a remedy for angina pectoris. In 1867 he published the results of his remarkable observations, and it is our privilege to reprint this classical contribution.

Brunton went to London in 1870 and was elected to membership in the Royal College of Physicians. He was appointed lecturer on materia medica and pharmacology at the Middlesex Hospital. A year later (1871) the post of casualty physician at St. Bartholomew's Hospital was vacated, and he was appointed to this office. That same year his wish to be associated in a teaching capacity with a famous medical school was fulfilled by his being elected joint-lecturer on materia medica and therapeutics at St. Bartholomew's Hospital. Until 1875 the lectures were divided with Dr. Frederick Farre and when Farre resigned, Brunton took undivided charge of them. That same year he was appointed assistant physician at St. Bartholomew's Hospital. In 1874 he became editor of "The Practitioner."

Under Brunton's direction, the whole scope of the lectures changed. He used physiology as the foundation for his teachings and based his results on experiments carried out on himself, on his pupils, and on animals. When he lectured, the theater was always crowded with students and with teachers from other schools. Brunton continued these lectures yearly as summer courses until 1901.

In 1874, at the early age of thirty, Brunton was elected a fellow of the Royal Society in recognition of his admirable work on the physiology of digestion and secretion, on the chemical composition of the blood, and on the action of digitalis and

mercury. He served as a member of the Council of the Royal Society from 1882 to 1884 and from 1905 to 1906, and was elected vice-president of the society the latter two years.

Brunton, in 1878, was placed in charge of the newly established department for diseases of the throat at St. Bartholomew's Hospital. He held this position until 1880, when he was succeeded by Sir Henry T. Butlin. From 1880 until 1895 Brunton was associated with the out-patient department of the same institution. In 1895 he became full-time physician at St. Bartholomew's Hospital, and he held that post until 1904. He resigned in 1904 because of illness and was immediately appointed a governor of the hospital and made an honorary consulting physician.

Brunton had a long and enviable professional career. For several years he served as examiner for the Royal College of Physicians and was a censor from 1894 to 1895. In 1877 he gave the Goulstonian lecture on "Pharmacology and Therapeutics." In 1889 he delivered the Croonian lectures on "The Connection between Chemical Constitution and Physiological Action." He was appointed Harveian orator in 1894, and he chose for his subject "Some Features in the Physiology and Pharmacology of the Circulation."

Among other appointments he was a member of the committee assigned in 1886 by the local government board to examine Pasteur's treatment for hydrophobia. In 1889 he was chosen by the editors of the "Lancet" to be a representative to repeat the experiments on the administration of chloroform that originally had been performed by members of a committee sponsored by His Highness, the Nizam of Hyderabad.

Besides holding membership in the chief medical societies of Great Britain, Brunton was a member of many foreign organizations. In the United States he was honored by the following societies: the American Academy of Arts and Sciences, the Academy of Natural Science of Philadelphia, the College of Physicians of Philadelphia, and the American Therapeutic Society. He was also a member of the Therapeutic Society of Paris and the Imperial Military Academy of Medicine of St. Petersburg.

He received the honor of knighthood in 1900 and was created a baronet in 1908. The Universities of Aberdeen and Edinburgh awarded him the honorary degree of Doctor of Laws, and the University of Dublin awarded him the honorary degree of Doctor of Science.

Brunton made numerous contributions to medical literature, many of which were of a pharmaceutic nature. From time to time he collected his writings and reprinted them in book form. In 1886 he published a volume "On Disorders of Digestion, their Consequences and Treatment"; in 1897 he published his "Lectures on the Actions of Medicine," and in 1901 appeared a collection of papers written between 1874 and 1901, called "Disorders of Assimilation."

In 1907 he published his "Collected Papers on Circulation and Respiration." Included in this group of his reprints is his classic on the use of amyl nitrite in the treatment of angina pectoris.

His most important work was his "Textbook of Pharmacology, Therapeutics, and Materia Medica." This volume appeared in 1885, and by 1887 three editions of the work had been published.

Brunton in 1879 married Louisa Jane Stopford, daughter of the venerable Edward A. Stopford, archdeacon of Meath. She died in 1909. His elder son, Major James Stopford Lauder Brunton, succeeded his father in the baronetcy on Sir Lauder's death in 1916. His second son, Dr. Henry Pollock Brunton, a member of the medical profession, was killed in action in France on October 8, 1915.

Sir Lauder died on September 16, 1916, and is buried in Highgate Cemetery in London.
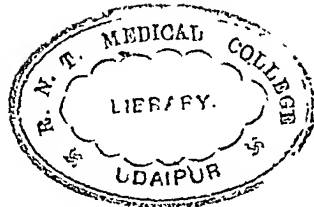
ON THE USE OF NITRITE OF AMYL IN ANGINA PECTORIS*

By

T. LAUDER BRUNTON B.Sc., M.B.

Senior President of the Royal Medical Society, and Resident Physician to the Clinical Wards of the Royal Infirmary, Edinburgh

FEW things are more distressing to a physician than to stand beside a suffering patient who is anxiously looking to him for that relief from pain which he feels himself utterly unable to afford. His sympathy for the sufferer, and the regret he feels for the impotence of his art, engrave the picture indelibly on his mind, and serve as a constant and urgent stimulus in his search after the causes of the pain, and the means by which it may be alleviated.

Perhaps there is no class of cases in which such occurrences as this take place so frequently as in some kinds of cardiac disease, in which angina pectoris forms at once the most prominent and the most painful and distressing symptom. This painful affection is defined by Dr. Walshe as a paroxysmal neurosis, in which the heart is essentially concerned, and the cases included in this definition may be divided into two classes.

In the first and most typical there is severe pain in the precordial region, often shooting up the neck and down the arms, accompanied by dyspnoea and a most distressing sense of impending dissolution. The occurrence and departure of the attack are both equally sudden, and its duration is only a few minutes.

In the second class, which from its greater frequency is probably the more important, though the pain and dyspnoea may both be very great, the occurrence of the attack is sometimes gradual, and its departure generally so; its duration is from a few minutes to an hour and a half or more, and the sense of impending dissolution is less marked or altogether absent.

Brandy, ether, chloroform, ammonia, and other stimulants have hitherto been chiefly relied upon for the relief of angina pectoris; but the alleviation which they produce is but slight, and the duration of the attack is but little affected by them.

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*Lancet 2: 97-98, 1867.

In now publishing a statement of the results which I have obtained in the treatment of angina pectoris by nitrite of amyl, I have to observe that the cases in which I employed this remarkable substance belonged rather to the second than the first of the classes above described.

Nitrite of amyl was discovered by Balard; and further investigated by Guthrie,* who noticed its property of causing flushing of the face, throbbing of the carotids, and acceleration of the heart's action, and proposed it as a resuscitative in drowning, suffocation, and protracted fainting.

Little attention, however, was paid to it for some years, till it was again taken up by Dr. B. W. Richardson, who found that it caused paralysis of the nerves from the periphery inwards, diminished the contractility of muscles, and caused dilatation of the capillaries, as seen in the web of the frog's foot.

Dr. Arthur Gamgee, in an unpublished series of experiments both with the sphygmograph and haemadynometer, has found that it greatly lessens the arterial tension both in animals and man; and it was these experiments—some of which I was fortunate enough to witness—which led me to try it in angina pectoris.

During the past winter there has been in the clinical wards one case in which the anginal pain was very severe, lasted from an hour to an hour and a half, and recurred every night, generally between two and four A.M.; besides several others in whom the affection, though present, was less frequent and less severe. Digitalis, aconite, and lobelia inflata were given in the intervals, without producing any benefit; and brandy and other diffusible stimulants during the fit produced little or no relief. When chloroform was given so as to produce partial stupefaction, it relieved the pain for the time; but whenever the senses again became clear, the pain was as bad as before. Small bleedings of three or four ounces, whether by cupping or venesection, were, however, always beneficial; the pain being completely absent for one night after the operation, but generally returning on the second. As I believed the relief produced by the bleeding to be due to the diminution it occasioned in the arterial tension, it occurred to me that a substance which possesses the power of lessening it in such an eminent degree as nitrite of amyl would probably produce the same effect, and might be repeated as often as necessary without detriment to the patient's health. On application to my friend Dr. Gamgee, he kindly furnished me with a supply of pure nitrite which he himself had made; and on proceeding to try it in the wards, with the sanction of the visiting physician, Dr. J. Hughes Bennett, my hopes were completely fulfilled. On pouring from five to ten drops of the nitrite on a cloth and giving it to the patient to inhale, the physiological action took place in from thirty to sixty seconds; and simultaneously with the flush-

*Journal of the Chemical Society, 1859.

ing of the face the pain completely disappeared, and generally did not return till its wonted time next night. Occasionally it began to return about five minutes after its first disappearance; but on giving a few drops more it again disappeared, and did not return. On a few occasions I have found that while the pain disappeared from every other part of the chest, it remained persistent at a spot about two inches to the inside of the right nipple, and the action of the remedy had to be kept up for several minutes before this completely subsided. In almost all other cases in which I have given it, as well as in those in which it has been tried by my friends, the pain has at once completely disappeared. In cases of aneurism, where the pain was constant, inhalation of the nitrite gave no relief, but where it was spasmodic or subject to occasional exacerbations it either completely removed or greatly relieved it. It may be as well to note that in those cases in which it failed, small bleedings were likewise useless.

From observations during the attack, and from an examination of numerous sphygmographic tracings taken while the patients were free from pain, while it was coming on, at its height, passing off under the influence of amyl, and again completely gone, I find that when the attack comes on gradually the pulse becomes smaller, and the arterial tension greater as the pain increases in severity. During the attack the breathing is quick, the pulse small and rapid, and the arterial tension high, owing, I believe, to contraction of the systemic capillaries. As the nitrite is inhaled the pulse becomes slower and fuller, the tension diminished, and the breathing less hurried. On those occasions when the pain returned after an interval of a few minutes, the pulse, though showing small tension, remained small in volume, and not till the volume as well as tension of the pulse became normal, did I feel sure that the pain would not return.

As patients who suffer from angina are apt to become plethoric, and greater relaxation of the vessels is then required before the tension is sufficiently lowered, I think it is advisable to take away a few ounces of blood every few weeks. When the remedy is used for a long time, the dose requires to be increased before the effect is produced. A less quantity is sufficient when it is used with a cone of blotting-paper, as recommended by Dr. Richardson, than when it is poured on a large cloth. From its power of paralysing both nerves and muscles, Dr. Richardson thinks it may prove useful in tetanus; and I believe that, by relaxing the spasm of the bronchial tubes, it might be very beneficial in spasmodic asthma. I have tried it in a case of epilepsy, but the duration of the fit seemed little affected by it. It produces relief in some kinds of headache, and in one of neuralgia of the scalp it relieved the severe shooting pain, though an aching feeling still remained.

While cholera was present in Edinburgh during last autumn, Dr. Gamgee proposed it as a remedy during the stage of collapse, a condition in which there are good grounds for supposing that the small arteries, both systemic and pulmonic, are in a state of great contraction. No well-marked case afterwards occurring in the town, he was deprived of an opportunity of putting it to the test, but it is a medicine well worthy of a trial, and should another epidemic unhappily occur it may prove our most valuable remedy.

Edinburgh, July, 1867.

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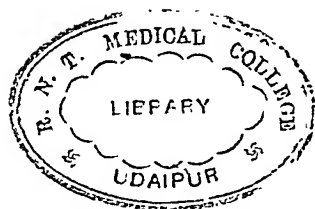
1868

HEINRICH QUINCKE

DESCRIPTION OF THE CAPILLARY AND VENOUS PULSE

HEINRICH QUINCKE
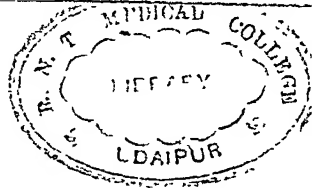
HEINRICH IRENAEUS QUINCKE

(1842-1922)

*H*EINRICH IRENAEUS QUINCKE was born on August 26, 1842, in Frankfort-on-the-Oder, Germany. He was the son of a well-known physician. His father moved to Berlin and there young Quincke attended the gymnasium. Quincke decided to prepare for a career in medicine after he had completed his academic training. He studied at the Universities of Berlin, Würzberg, and Heidelberg. His teachers included the anatomist, Heinrich Müller; the Swiss histologist, Albert von Kölliker (under whom he published a paper on the ovaries in the mammal); the physiologist, Henry L. F. von Helmholz; the chemist, Robert W. E. von Bunsen; and the master pathologist, Rudolf Virchow.

In 1863 he received his medical degree from the University of Berlin, and in 1865 he worked under Ernst Wilhelm von Brücke in physiology in Vienna. Quincke then spent a period of observation in the hospital and in the clinics of Switzerland, Holland, France and England.

He returned to Germany where he became Friedrich Theodor Frerichs' assistant in Berlin. At the age of thirty he succeeded Bernard Naunyn as professor of medicine at the University of Berne, in Switzerland. Five years later he accepted the professorship of medicine at the University of Kiel in Germany, where he continued to work until his retirement in 1908. He then moved to Frankfort-on-the-Main and during the First World War was still active as a practicing physician. He died in May of 1922 at the age of seventy-nine. Death came to him suddenly and quietly as he was seated at his writing table at his home in Frankfort.

Quincke was interested throughout his long career chiefly in the problems of general pathology and physiology, and he made many important contributions to these fields of medicine, in addition to his contributions to neurology for which he is so well known.

He was the first physician to study, in detail, the capillary and venous pulsations and to evaluate their significance in establishing the diagnosis of aortic insufficiency. He made this study in 1866, and it is a pleasure to present to our readers in translation the classic account of his work which he published in 1868. In 1870 he published his observations on aneurysm of the hepatic artery. In 1876 he contributed the section on the diseases of the arteries for the "Handbuch der speciellen Pathologie und Therapie" of Hugo Wilhelm von Ziemssen.

Two outstanding contributions of clinical importance were published by Quincke which in their day did not attract notice. The first of these appeared in 1875 in the "Berliner Klinische Wochenschrift," and was entitled "Über Vagusreizung beim Menschen." Therein he called attention to a phenomenon which today is of the utmost clinical value, namely, the observation that pressure on the carotid artery of the neck results in a slowing of the pulse and he explained that stimulation of the vagus nerve was the cause of this phenomenon.

With Heinrich Hochhaus in 1894 in the "Deutsches Archiv für klinische Medizin" he published a paper entitled "Über frustrane Herzkrankheiten." Therein Quincke coined the term "Frustraner Kontraktion" of the heart to explain the extrasystoles

in which the heart sounds are heard but no peripheral pulse can be felt. In 1882 Eugen Dinkelacker published at Kiel a 27-page monograph containing an observation of scientific importance which he had noticed in the clinic of his chief, Heinrich Quincke; the monograph was called "Über acutes Oedem." Quincke later described this observation in more detail. The condition is known as "Quincke's edema," and he was able to show its association with urticaria and erythema multiforme.

Quincke's greatest contribution to medicine probably was his invention of a diagnostic procedure, the lumbar puncture.[1] There can be no doubt that this technique contributed as much to medical advancement as did Auenbrugger's discovery of percussion and Laënnec's development of auscultation. Quincke not only emphasized the diagnostic value of the spinal puncture but also pointed out its therapeutic significance.

Quincke made other observations in neurology which made him famous in this field. In 1909-1910 he published his studies[2] on the pressure of the spinal fluid and discussed in detail diagnosis by examinations of the cells, chemical observations or reactions, and the immune biologic properties of the spinal fluid. His interest in the physiology of the spinal fluid dated back to 1872, in which year he described the anatomic relationship between the arachnoid and subarachnoid space, the relationship of the optic nerve to the cranial cavity, the function of the choroid plexus, and studies on the ventricles of the brain.

A most important contribution to neurology was Quincke's description of "meningitis serosa," a contribution first published in 1909.[3] Quincke made many other contributions to neurology, too numerous to mention.

In the field of hematology, his studies concerning pernicious anemia, destruction of the erythrocytes, and the production of hemosiderosis were among the first published accounts and contributed much to modern understanding of the physiology of hemoglobin metabolism.

Quincke, in his later years, described the connection between hemolysis of the erythrocytes and the formation of bilirubin. On the basis of clinical and experimental data he made some important contributions to the problem of jaundice.

It seems unbelievable that one man was able, in the span of his life, to make so many important contributions to medicine. But Quincke made many more observations than those thus far mentioned. He was probably the first to treat abscesses of the lung surgically, and in 1901 published a fundamental paper on surgery in the treatment of tuberculosis. Furthermore, he contributed important papers on the treatment of diabetic coma, Addison's disease, amoebic dysentery, typhoid fever, peptic ulcer, and postural drainage in the presence of abscesses of the lung.

These are but a few of the outstanding and original contributions which modern medicine owes to the great clinician and physiologist, Heinrich Quincke.

[1] Dr. Essex Wynter of England discovered lumbar puncture, independently, about this time.

[2] Published in Zeitschrift für Nervenheilkunde.

[3] Quincke, Heinrich: Zur Pathologie der Meningen, Deutsche Ztschr. f. Nervenh. 38: 343-399, 1909.

II. OBSERVATIONS ON CAPILLARY AND VENOUS PULSE*

By

DR. H. QUINCKE

Assistant in Medicine at the University Clinic of Berlin

IT IS an acknowledged fact in physiology that the pulsations in the arterial system which arise in the heart, extend to the smallest arteries, and that the blood stream in the capillaries is not influenced in the same manner by the beat of the heart. This belief is chiefly based on microscopic observations of the capillary stream in the mesentery of various animals, in the web membrane of the frog and of the bat. Only when there is a diminished flow of blood in the veins, or when there is a marked reduction of the arterial pressure and slowing of the heart beat, such as occurs in the dying animal, is a propulsive movement of the blood in the capillaries observed.

Only one such observation in man exists, that of Lebert (*Handbuch der practischen medizin* I., p. 725), who observed in a case of aneurysm of the aorta, systolic reddening and diastolic blanching of the cheeks, constituting an actual capillary pulse.

Cl. Bernard observed a transmission of the pulse through the capillary system to the vessels of the submaxillary gland, extending to the tributary veins, when, following section of the sympathetic, simultaneous stimulation of the lingual branch produced maximum dilatation of the arteries.

Pertaining to man, only a few observations on the venous pulse exist, those by King (*Guy's Hospital Reports* IV, XII) cited briefly by Stokes (*Diseases of the Heart*). The original, unfortunately, has not been available to me.

There are, however, places in the human body, where under completely normal conditions, but more clearly under pathologic conditions, one frequently observes the transmission of the pulse wave from the heart reaching to the capillaries and then into the veins: these sites are the fingernails, hand, forearm and foot.

Insofar as the capillary pulse is concerned, it can be observed in one's own fingernails, but still clearer in those of another, in the region between the whiter, anemic area and the redder, more injected part of the capillary system of the nail bed; in the majority of persons examined,

there occurs, synchronous with the beat of the heart, a to-and-fro movement of the margin between the red and the white portion, and one can be convinced that the exaggeration of the redness ensues a moment after the apex beat and is still definitely systolic and occurs rather rapidly, while the recessive movement of the reddened margin occurs more slowly, that is, a perceptible delay in the wave as seen by the eye, in the same manner that palpation and the sphygmograph reveal it in the pulse waves of the radial artery.

But the fingernails of everyone do not show this aforementioned white zone; in plethoric persons with strong and frequent heart action and high arterial pressure, in warm outside air the nails are not uniformly reddened. Under these circumstances a clearer zone in the nail can be produced by (naturally similar) pressure, or better, by elevating the arms; by the latter manipulation one has at the same time the advantage that the blood pressure in the vessels of the arms falls, and the increased pressure (with constant cardiac action) which the blood derives with every beat of the heart is greater than the average pressure existing when the hand is not elevated.

Similar conditions, which are produced by elevating the hands, that is, increase of the anemic areas of the nails and lowering of the arterial pressure, commonly occur in anemic individuals: therefore, in those the capillary pulse is usually more distinct and also visible without elevating the arm: but, the activity of the heart must not be greatly diminished, and therefore it is most distinct in mild chlorotics and not in convalescents following severe diseases.

Greater amplitude and great output of individual heart contractions favor the distinctness of the capillary pulse, while marked increase in the arterial tension and greater frequency of heart contractions are less favorable. The capillary pulse will either be perceived more distinctly or (more frequently) less distinctly, according to the predominance of one or the other factor in fever and under excitement. The presence of large and rapidly falling pulsations is demonstrated in an exquisite manner in insufficiency of the aortic valves; for that reason the capillary pulse is especially distinct. Even in the horizontal position of the hand one observes a distinct and rapid to-and-fro movement of the margin between the red and white zones, and also with a uniform coloration of the nail and a lightning-like and momentary accentuation of the reddening, so that the mannner of the appearance and disappearance of the capillary pulse, is objectively as characteristic a sign of aortic insufficiency as the exquisitely abrupt pulse is to the palpating finger. Surely it is not so constant, for here likewise, in the appearance of the capillary pulse under normal conditions, the necessary conditions are not always present, and the requisite softness and transparency of the nail and the appropriate degree of elasticity of the arterial system must be considered.

It is, generally, impossible to say in which fingernail the phenomenon is most distinct, but it seems to occur most frequently in the index finger. The white zone is usually found in the third quarter of the nail, measuring from the matrix, and the pulsation is at times more distinct in the lower part, at other times in the upper part.

Up to the present time I have not been able to observe the capillary pulse in the toenail; the reason probably being the diminution of the pulse wave in the long arterial tube and the great hardness and thickness of the nails.

Noteworthy is the observation of Kölliker (*Gewebelehre*, pp. 121 *und* 580) that in the production of the capillary pulse in the nail bed the average diameter of the capillaries is 0.005-0.008, while elsewhere in the human body it is only 0.002-0.006.

I have been able only a few times recently to observe a propagation of the pulse wave through the capillaries into the veins in individuals without valvular insufficiency.

The first object of observation was my own hand. Here, I saw on several occasions, after the veins on the back of my hand were markedly dilated from great heat, a weak but unquestionable post systolic pulsation.

Much more distinct was the venous pulse in a 50-year-old woman, H., who entered the hospital owing to cholelithiasis. She had never complained of palpitation, the heart sounds were clear; there was perhaps slight hypertrophy of the left ventricle, but this could not be definitely determined, owing to the unusual rigidity of the thorax. The palpable arteries were somewhat rigid, the pulse full, quite resistant, the pulse waves not especially short but waning abruptly. An extraordinarily distinct pulse was visible in the distended and prominent veins of the very thin and redundant skin of the back of the hand and forearm, as well as in the cross anastomoses extending up to the middle of the forearms, which even appeared delayed as compared to the same radial pulse, and still more evident as compared to the carotid pulse. The capillary pulse in the nails, in this case, was furthermore very distinct.

A third case of venous pulse which first came under my observation during the last few surviving days of life, occurred in a robust young man, who while diving in water, struck his head on the bottom and suffered a paralysis of all the spinal nerves issuing below the fourth cervical vertebra (the necropsy revealed a fracture of the vertebra, with crushing of the cord). In the veins of the back of the hand extending to the middle of the forearm, as in the foregoing case, a post-systolic pulsation was distinctly observed. It is highly probable that here we were dealing with a paralysis of the vasomotor nerves, which together with the high temperature of the air, produced dilatation of the vessels resulting

most distinct at the middle in the border of the physiologic cupping; whereas in the finger nails, the border between red and lighter red moved to and fro with the pulse. The capillary pulse was not visible beyond the border of the papilla, even when present, because here the color change, owing to the dark background of the choroid, was not sharply differentiated. Furthermore, neither eye revealed any other abnormality accounting for the phenomenon of pulsation.

The diastolic pulse in the central vein of the retina, which is frequently normally present, was seen with great intensity; since the production of this pulsation has not been entirely explained, and is probably not the result of transmission through the capillaries, it will not be discussed further at this time.

From the above observation, it is apparent that the pulse wave which originates in the left side of the heart, is not as extensive as generally believed and disappears in the small arteries.

Under appropriate conditions, only partly pathologic, partly within the limits of normal, the wave is transmitted through the capillaries, even into the veins, and it is also probable that the capillary pulse can be observed in other places than those mentioned; for example, internal viscera rich in blood, such as the spleen and kidneys. The hands are a good place for observing it, partly because of the superficial situation of the capillary and venous network and also because the peripheral areas have an extensive blood supply and relatively broad arterial tributaries; the marked change in volume when hot or cold, reveals how extraordinarily adequate the blood supply is.

I wish to express my best thanks to *Geheimrath* Frerichs for his generosity in making available the material in his clinic for these observations.
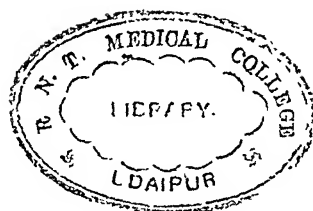
1870

SAMUEL WILKS

DESCRIPTION OF BACTERIAL ENDOCARDITIS

SIR SAMUEL WILKS

(Courtesy Annals of Medical History.)
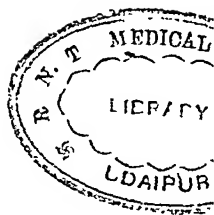
SIR SAMUEL WILKS

(1824-1911)

*"Of all the boys that are so smart,
There's none so smart as Sammy;
He is the darling of our Art,
And Guy's his Alma Mammy."*

—W. L. Braddon, quoted by W. H. White,
Guy's Hospital Reports, 1913.

ON JUNE 2, 1824, Samuel Wilks was born at Camberwell, London, England, the second son of Joseph Barber Wilks, who was a cashier of the East India House. Young Wilks began his education in a dame's school. Later he attended a boy's school at Camberwell Green. At the age of eleven he was tutored by the Reverend Dr. Spyers at Wallop. When Dr. Spyers was made head master at Aldenham, Wilks accordingly went to Aldenham, where he stayed for three years. In 1839 he spent one year at University College School, and in 1840 was apprenticed to Mr. Richard Prior, the family physician, of Newington.

During the second year of his apprenticeship he attended the course on anatomy given under the direction of Mr. Bransby Cooper at Guy's Hospital. The next year he continued the study of medicine at Guy's Hospital. When he completed his qualifications he passed the examinations for a diploma from the College of Surgeons and the Apothecaries Hall.

Wilks decided to qualify for a medical degree and therefore matriculated at the University of London. When he completed his classical course, he studied medicine at this institution. In 1848 he received the degree of Bachelor of Medicine and in 1850 the degree of Doctor of Medicine. For his high scholastic average in obtaining the latter degree he received the gold medal.

In 1851 Wilks became a member of the Royal College of Physicians, and in 1853 he was appointed physician to the Surrey Dispensary. He was married in 1854 to Mrs. Prior, the widow of Richard Prior under whom Wilks had served his apprenticeship.

The appointment of Wilks as assistant physician to Guy's Hospital took place in 1856. At that time Richard Bright (1789-1858) was consulting physician and Thomas Addison (1793-1860) was one of the physicians on the staff. That year, also, Wilks was elected a fellow of the Royal College of Physicians. At Guy's Hospital, Wilks served as pathologist, making systematic post-mortem examinations. He also lectured on pathology and was curator of the museum. He became full physician in 1867.

In 1859 Wilks published the first edition of his "Lectures on Pathological Anatomy." He did much to place the study of pathology on a scientific basis in England. Formerly, physicians often had performed necropsies in order to ascertain whether or not their diagnoses had been correct; Wilks encouraged physicians to conduct examinations routinely to learn more about disease.

In 1863 a paper was published by Wilks in "Guy's Hospital Reports" entitled: "On the Syphilitic Affections of Internal Organs." According to White, Wilks was

the first to notice that syphilis, in some cases, attacks the viscera. Possibly as a result of that observation, he was elected a fellow of the Royal Society in 1870.

Wilks was one of the first to report on what he called "arterial pyaemia," now known as "bacterial endocarditis." We are reproducing his classic description of that condition, originally published in 1870 in "Guy's Hospital Reports."

Wilks soon began to devote more of his time to study of the diseases of the nervous system. His most original work in this field was published in the "Lancet" in 1872. It was a study of alcoholic paraplegia. He was mistaken in attributing this disorder to changes in the spinal cord, but he was the first to associate abuse of the use of alcohol with a form of paraplegia. Wilks published his comprehensive study, "Lectures on Diseases of the Nervous System," in 1878. A second edition of the book was published in 1883.

In 1879, Wilks was appointed physician to the Duke and Duchess of Connaught. That same year he delivered the Harveian Oration before the Royal College of Physicians.

Wilks was an active member of the British Medical Association. In 1872 he delivered the address in medicine at the annual meeting which that year was held in Birmingham. He was president of the Section of Medicine at the meeting held in Cardiff in 1885; and in 1895, at the meeting held in London, he was president of the Section on Pathology and Bacteriology.

In 1881, Wilks delivered an address before the Pathological Section of the International Medical Congress, which was held in London. From 1881 to 1883 he was president of the Pathological Society.

In 1884, Wilks received the honorary degree of Doctor of Laws from the University of Edinburgh. In 1885 he was made a member of the Senate of the University of London. That same year, at the age of sixty-one, he retired from the active staff of Guy's Hospital and was made a consultant.

The Royal College of Physicians in 1896 elected him to the presidency of that organization. A year later he was created a baronet on the occasion of the Diamond Jubilee celebration of Queen Victoria. He also was made physician extraordinary to the Queen.

Sir Samuel retired from active practice in 1901, at the age of seventy-seven. He spent the remainder of his life in Hampstead, where he endured several severe illnesses. He suffered from an inflamed appendix, which was removed. He later was operated on for removal of an enlarged prostate gland. He also suffered from a cerebral attack accompanied by unconsciousness which lasted two or three days. This, in turn, was followed by paraplegia which confined him to bed for many months and prevented motion of the lower limbs. In spite of his ill health, at the age of eighty-five he began to write his "Memoirs."[1] This work was finished and the proof was corrected by the time he reached the age of eighty-seven. Wilks finally lay in bed completely paraplegic. He died on November 8, 1911, and was cremated on November 11.

[1]Wilks, Sir Samuel: A memoir by Sir Samuel Wilks on the new discoveries or new observations made during the time he was a teacher at Guy's Hospital, London, 1911, Adlard and Son, 200 pp.

CAPILLARY EMBOLISM OR ARTERIAL PYAEMIA*

By

SAMUEL WILKS

DID space allow I could report several cases of this disease exemplifying the constitutional symptoms attendant upon the passage of disintegrating fibrin through the system, but I will content myself with reminding the reader of the importance and frequency of the complaint, although it is one which is constantly overlooked. It may be safely said that there has been no more important addition to pathological science than the doctrine of embolism; the facts included in it have been at once recognised by the profession as throwing a light upon cases which before were most obscure. It is remarkable, however, that only one portion of the statements in Dr. Kirkes' paper seems to have been generally apprehended by medical men,—that portion which alludes to the effects of the blocking of a large vessel by a vegetation carried from the heart: such as the plugging of a cerebral vessel, and the attendant paralysis; or the plugging of an artery in a limb, and the consequent gangrene. But there is another, and equally important, part of this paper in which he speaks of the blocking of the smaller vessels in the parenchymatous organs, with constitutional symptoms. The results seen in the organs have long been known and described by Rokitansky under the term capillary phlebitis, but the severe and even fatal symptoms often attendant thereon have not yet been sufficiently recognised. By the term embolism, I say, is generally implied the case of the blocking of a large vessel and the resulting local symptoms, but under it should be included the equally important and common case of the obstruction of the smaller arteries; with attendant constitutional symptoms. I have for many years been in the habit of insisting upon this both in the post-mortem room and in the wards. Formerly, I was accustomed to show from a strictly pathological point of view how changes occurred in the arterial system analogous to those which take place in the venous:—that as in phlebitis some morbid matters, products of inflammation, being taken up by a vein and carried inwards through the circulation, give rise in the internal organs to depositions of a similar kind in them, so in the arterial system disintegrating fibrin of the blood may be carried from the centre of the circulation to the periphery, and there give rise to further deposits of a like fibrinous matter. Latterly, I have been enabled to show clinically that in both

cases there are attendant febrile symptoms with characteristic arthritic pains and occasional rigors; and that just as there is a venous pyaemia having its source on the outside of the body, so there is an arterial pyaemia having its origin within. The term "pyaemia" is, of course, not used in its strict etymological sense; but then it must be remembered, that a rigid application of the word is not required in the more ordinary case of contamination of the venous blood; it is for the objectors to use the term "septicaemia."

This form of affection, I believe, is far from uncommon, as pointed out in a lecture, of which an extract is given in *British Med. Journal* of March, 1868. That it is overlooked arises from the circumstance that a severe organic disease exists generally at the same time, and that this is considered sufficient to account for the symptoms and death. A patient, for example, is in hospital for valvular disease of the heart arising, perhaps, from rheumatism at some former period; whilst under observation he may have febrile attacks attended by articular pains, but these are regarded as touches of the primary complaint, and when after death the valves are seen covered with vegetations, and the spleen and kidneys full of softening fibrinous masses, these are in no wise regarded as having been instrumental to the fatal event, but as mere accidents of the disease. It must, however, have often, I believe, occurred to the observer that the derangement of the valvular apparatus was scarcely sufficient to cause death. I can myself recall more than one instance where a patient died several weeks after an endocarditis, and the cause was attributed to heart disease, but where the valves were apparently quite efficient. The cases above all others which afford the most striking examples of arterial pyaemia are those where the endocarditis has left the valves of the heart altogether structurally uninjured, and, therefore, where no mechanical causes resulting from heart derangement can possibly have produced death. Such an instance I mentioned in the lecture above referred to, where a medical man was seized with all the symptoms of pyaemia, in so marked a degree that the only question discussed by his attendants was the probable source of infection. This turned out not to be in the veins at all, but in the arterial system, where the origin of the disintegrating fibrin was found to be an aneurism in the auricle of the heart. Such a case was a simple one of death by arterial pyaemia without any organic lesion. Of course, this constitutional affection may be seen in conjunction with that of local embolism, as in a case I took to the Pathological Society, where a man, besides having in the profunda artery of the leg an embolus threatening gangrene, had articular pains in all the limbs, with febrile symptoms dependent on the circulation of smaller particles of deleterious matter through the system.

The occurrence of fibrinous masses in the solid organs of the body has long been known, and as early as the year 1832 a kidney thus affected

is portrayed in these Reports. Rokitansky described the condition under the name of capillary phlebitis, and states his opinion that it is due to some spontaneous disease of the blood. It was mainly to refute this that Dr. Kirkes published his paper, in which he proved that particles of fibrin were carried to a distance from the heart, where they had previously been formed. This constituted Kirkes' originality. I have, however, never given up the opinion that in many cases the deposit may have occurred from a primary change in the blood itself, since often there is no proof of the existence of a primary endocarditis; in fact, there has been reason to believe that in some cases the deposit found on the valves of the heart has occurred simultaneously with the deposits in the solid viscera. It must be admitted that if, on post-mortem examination, there be found associated with these deposits in the viscera some vegetations on the cardiac valves, it would be presumptuous to deny that an endocarditis might have been the origin of the whole train of subsequent events; yet, on the other hand, it would be a practical error not to be awake to the possible occurrence of arterial pyaemia, because there is no history of a primary cardiac affection. For my own part, although Kirkes may be right in the main, I think there is every reason to believe that Rokitansky's statement is equally true, that deposits may occur from changes in the blood itself. Practically I am sure the supposition of such an event will enable us to recognise cases otherwise obscure.

The facts, then, are these—there is the simple case of endocarditis, or the case where vegetations are covering chronically diseased valves, and as a result the deposition of fibrinous material in the capillaries of organs and other parts of the body. In such a case the symptoms and death by arterial pyaemia are, I believe, frequent enough, although not so generally recognised as they should be. I would also insist that, irrespective of a history of a primary heart affection, such symptoms of pyaemia should lead us to a careful examination of the heart in all cases, when the existence of a bruit may at once suggest their true nature. Whether in such a case the vegetations on the valves which are productive of the morbid sound existed previous to the formation of deposits elsewhere, and were the source of them, or whether they occurred simultaneously from a blood-change, is a question often as difficult to decide after death as before it. Then, again, we may meet with cases where the patients present all the symptoms of pyaemia, and where, failing to find any source for the blood infection on the surface of the body to contaminate the venous blood, we may conjecture that the pathological processes are going on in the arterial system, even though we fail to detect any morbid sounds in the heart; for a post-mortem examination sometimes shows that the viscera are affected in the manner above stated, when the interior of the heart is altogether healthy.

One reason I have for believing that in many instances the source of infection is not the carrying away of vegetations from the heart is, that in the obscurer kind of cases the effects are very slow in developing, whereas in the instances where large portions of fibrin are carried away in the stream of blood the effects are more sudden and momentous. In these chronic cases the disintegration of the fibrin is slower, the smaller vessels in the viscera are occluded, and the organs which suffer are other than those most usually selected in marked heart disease. Thus the spleen not only has isolated masses within it, but the whole organ becomes enlarged by the deposition throughout it. In the same way the liver may be enlarged and hardened, and also the lungs. If in such a case the disease of the blood were due, not to some morbid process originating in the fluid itself, but to a change wrought upon it by the lining membrane of the heart, I should conceive that the latter, although in a sufficiently unhealthy state to be competent to effect this change, was not covered with vegetations, so as to give rise to a bruit, or to account directly for the deposits in the organs by simple transmission. In time, of course, such vegetations might arise, and produce a murmur.

In these cases it is probable that the first symptoms which attract attention will be the constitutional ones, and that febrile symptoms and occasional rigors will suggest the existence of ague; in fact I have seen several cases of pyaemia, both venous and arterial, treated for miasmatic fever. After a short time it is possible that the liver and spleen may be felt enlarged, and still the obscurity remain. At a later period a bruit may be heard, which may be styled aortic or mitral, according to position. The febrile symptoms continue, and the patient, perhaps after a protracted illness, dies; the organs are found affected as described, and vegetations on the valves of the heart. There may be appearances suggesting an old cardiac disease, and accounting for the deposition of fibrin; but it is equally probable that there may be nothing in the heart to indicate an older change than that observable in the solid viscera. I have now seen so many instances of this, both in hospital and private practice, that I recognise them as belonging to a class, although I am often unable to state the origin of the blood change.

That endocarditis with vegetations on the valves is not necessarily a primary affection is seen in the fact that it may result from an ordinary venous pyaemia; for example, a healthy man may fracture his leg so as to necessitate its amputation; after a time he may have pyaemia, and then an endocarditis. In scarlatina, after the usual recovery, a secondary fever of the nature of a pyaemia is often seen, in which pains in the joints occur, and not unfrequently an endocarditis, so that it is not uncommon for an organic disease of the heart to be traced back to an attack of scarlatina. More than this, in post-mortem examinations of children

who have died of this disease, and even before the accession of well-marked pyaemic symptoms, fibrinous masses may be found in the spleen and kidneys.

During the last few years, since my attention has been drawn to the subject, I have seen many instances of arterial pyaemia, and my mind reverts to cases occurring at an earlier period, and not at all explicable by the pathological doctrines of that day. In a paper on Pyaemia in Vol. 7 of the present series of these Reports, I relate a few cases where there was no evidence that endocarditis was the primary affection. One was the case of a man who was admitted after discharge from prison; he was extremely low and depressed, and thought to be suffering from fever. He shortly died, when the viscera were found full of masses of softening fibrin, and a vegetation existed on an aortic valve, but when this was removed the endocardium showed no evidence of inflammation. Another case of Dr. Habershon's, which had previously been reported by him in Vol. 5, was that of a woman in a state of extreme poverty. Being very ill she was sent to the hospital as a case of fever; the febrile symptoms were high, the tongue brown, the pulse quick, and there were daily rigors, followed by heat and sweating; no cardiac bruit could be heard. She remained exceedingly ill, and was in that state which is usually called typhoid, when some blebs came out on the skin, and she had pains in all the joints and the rigors continued. On the third day after admission a systolic murmur was heard, she gradually grew more prostrate, became delirious and died. On post-mortem examination the spleen and kidneys were found full of softening masses of fibrin, and there was a slight roughness on the border of the mitral valve, as if vegetations might have once existed there. In some cases, as I have said, the liver and spleen may be enlarged, and thus the observer's whole attention may be given to the abdomen. I well remember how such a case, when I was a pupil, puzzled Dr. Addison. A woman for three months had had fever with rigors, supposed to be ague, and, with this, enlargement of the liver and spleen. After death the heart was found diseased, although no evidence was given of it during life. In looking through the "Transactions of the Pathological Society," I have no doubt that several cases of enlarged spleen were of the nature indicated, and as early as the year 1851, about the time when Dr. Kirkes was engaged in his researches, a case was brought to the Society by Dr. Hare of a young man, aet. 25, who gave as his history that he took cold six weeks before, and that this was followed by pains in the joints, palpitation, and enlargement of the abdomen. When under care he had a large liver, a large spleen, and albuminous urine; also oedema of the ankles, and a double murmur over the cardiac valves. After death there were found vegetations on the aortic and mitral valves; the spleen and kidneys were much enlarged, hard, elastic, with a yellowish deposit.

I would say, therefore, that arterial pyaemia is a by no means uncommon affection, and that it is seen frequently in chronic heart disease; but the symptoms are overshadowed by the more severe ones attendant on the valvular imperfection, or, if observed, regarded merely as rheumatic. Also, that it may be often met with where there is no history of a primary heart affection, although an endocarditis at the time of the occurrence of the symptoms may exist. Also that it should be suspected in cases of obscure febrile conditions, especially if accompanied by rigors, and more especially where the liver and spleen have been found to be slowly increasing in size.
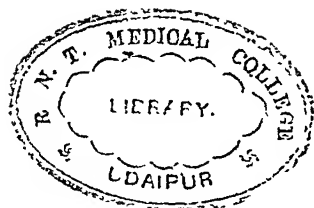
1872

LUDWIG TRAUBE

DESCRIPTION OF PULSUS ALTERNANS

LUDWIG TRAUBE
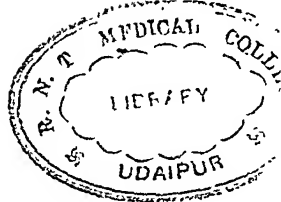
LUDWIG TRAUBE

(1818-1876)

*"Today, also, I maintain that experiment is the 'sine qua non' of scientific
pathology. . . . Even therapeutics, I am convinced, will take a definite step forward
when an attempt is made in a systematic way to modify the disease processes produced
in animals by the well known drugs."*

—Ludwig Traube, in Introduction to his
Collected Works.

LUDWIG TRAUBE was born on January 12, 1818, in Ratibor in Silesia, Germany.
He received his academic training at the gymnasium in Ratibor, and at the
age of seventeen he matriculated at the University of Breslau. It was his father's
wish that young Traube should become a physician, so he began the study of medi-
cine at Breslau under the Bohemian physiologist, Johannes Purkinje (1787-1869),
who at that time was the leader of medical thought in Breslau.

Two years later, Traube went to Berlin, where he was attracted by the magnetic
personality of Johannes Müller (1801-1858). It is said that the general state of
medical education in Germany at that time was low and Traube, being greatly disap-
pointed, decided to give up his medical career. His father, however, insisted that his
son continue his medical studies, which the young man did. He devoted as much
time as possible to the study of French medicine, which during this period was out-
standing because of the teachings of François Magendie (1783-1855) and René
Laënnec (1781-1826).

In 1840 Johann Lukas Schönlein (1793-1864) came to Berlin. He greatly im-
pressed Traube, who later (1849) was to become Schönlein's assistant. Meanwhile,
Traube decided to study at Vienna where Karl von Rokitansky (1804-1878), the
famous pathologist, and Josef Skoda (1805-1881), who introduced Laënnec's method
of auscultation into the medical curriculum, were his teachers. Traube received the
degree of Doctor of Medicine in 1841 but remained in Vienna for some time as a
graduate student.

Traube later returned to Berlin to practice medicine. He had decided to devote
himself to scientific research, but met with much opposition from various persons.
At that time the Charité was the only public hospital in Berlin. It was exclusively
controlled by the army, and in order to use the hospital Traube was obliged to be-
come an assistant to an army physician. He practiced medicine in a suburb of
Berlin, and being accustomed to study cases that demanded scientific investigation,
he frequently was obliged to pay for permission to perform a necropsy.

Even though they were in many ways deprived of clinical resources, Traube and
other young physicians met regularly once a week to discuss and read the outstand-
ing researches of Magendie and Claude Bernard (1813-1878).

Soon Traube's ability and skill in the newer diagnostic methods of auscultation
and percussion were manifested and many young medical students requested him
to lecture to them in private courses. Although his pupils were enthusiastic about
their teacher, patients were annoyed by being subjected to too many examinations

[587]

and a regulation was passed by the Charité, directed against Traube, which stated that army physicians were required to do their own work without the help of assistants.

Being unable to continue his clinical studies at the bedside of patients at the Charité, Traube, greatly influenced by the famous experimental studies of the French physiologists, decided to turn to the exact methods of animal experimentation. After two years of study he published his first paper, "On the Causes and Origin of Those Changes Which the Lung Parenchyma Suffers After Section of the Vagi." This classic work brought him into contact with Rudolf Virchow (1821-1902), who was then assistant pathologist at the Charité. A lifelong friendship began.

In 1846 the first number of a journal originated by three friends (Traube, Benno Reinhardt, and Virchow) was published. It was called: "Beiträge für experimentelle Pathologie und Physiologie," and was superseded in 1847 by "Virchow's Archiv." This event marked a revolutionary milestone in German medicine. In his introduction to the first issue, Traube demanded a different approach to the study of scientific medicine, an approach based on experimentation and its correlation with clinical experience. In the second number of this journal (1847), appeared the famous paper by Virchow on the occlusion of the pulmonary artery and the consequences thereof. Traube also contributed his classical account on suffocation to that issue of the publication.

Traube's primary aim—to contribute to the advancement of modern clinical medicine—was not fulfilled, however, until 1849, when Virchow made it possible for him to receive a hospital appointment. At that time he became an assistant to Schönlein. In 1857 he became an associate in the Charité and was appointed an assistant professor at the University of Berlin, where his reputation as an outstanding physician soon became known. At the University of Berlin he became the leading teacher, but not until 1872 did he receive his full professorship.

During the years previous to his association with the University of Berlin, Traube experienced a period of great disappointment and several times was tempted to ask for his release. It was a battle against the political factions of the state directed against Traube mainly because he was a Jew. Only the personal friendship of sincere colleagues gave him enough moral support to withstand repeated attacks.

Traube's scientific contributions are contained in three volumes.[1] The first includes his physiologic studies and the second and third his clinical and pathologic researches.

Among the different papers in the first volume his studies on asphyxiation and his pharmacologic researches on the action of digitalis, curare, nicotine, bile salts, and potassium nitrate, must be mentioned.

The variety of outstanding contributions Traube made in clinical and pathologic research as reflected by the last two volumes of his collected works is remarkable. His studies on diagnostic procedures in relationship to diseases of the chest (Traube's semilunar space and Traube's double sound) have become associated with his name. These volumes, too, contain his important studies on bronchitis, abscess, and gangrene of the lung and the invasion of the lungs by particles of coal.

In the field of cardiology, two of Traube's papers are outstanding. In 1856 he published a comprehensive account, "Über den Zusammenhang von Herz und Nieren Krankheiten," in which he discussed renal lesions caused by passive congestion of cardiac origin. He separated this condition from the inflammatory group of kidney lesions observed in the presence of Bright's disease. In the second part of this paper Traube described the symptomatology of contracted kidney and explained

[1]Traube, Ludwig: *Gesammelte Beiträge zur Pathologie und Physiologie,* Berlin, 1871-1878, A. Hirschwald, 3 v. in 4.

the associated cardiac hypertrophy as being the result of increased circulation observed by the diminution of renal capillaries.

In 1871 Traube read his classic paper on "A Case of Pulsus Bigeminus" before the Berlin Medical Society. It is our privilege to present this report to the reader in translation. The report contains the first clear picture of pulsus alternans.

Many honors came to Traube late in life as acknowledgment of his great scientific contributions in the field of medicine. But whatever the honor paid him, equally great in his regard was the admiration of his pupils and associates who saw in him one of the foremost physicians of the time.

At the age of fifty-six, Traube suffered, as so many physicians before and after him have suffered, repeated attacks of angina pectoris. Finally congestive heart failure developed, to which disease he succumbed in April of 1876 at the age of fifty-eight.

Traube, a true scientist, diagnosed his own disease as follows: "Hypertrophy and dilatation of both ventricles with arteriosclerosis and coronary sclerosis with partial fatty degeneration of the myocardia."

A CASE OF PULSUS BIGEMINUS, INCLUDING REMARKS ON THE ENLARGEMENT OF THE LIVER IN VALVULAR INSUFFICIENCY AND ON ACUTE ATROPHY OF THE LIVER*

By

PROF. DR. L. TRAUBE

IN MY experiments on animals I have for years been acquainted with a type of pulse, which I have named "pulsus bigeminus." The observations relative to this are unfortunately somewhat scattered, but are available in my collected "Contributions to Pathology and Physiology."

The nature of the pulsus bigeminus may be said to be this: following every two pulses which originate in the aorta, a longer pause ensues. This phenomenon is differentiated from the pulsus dicroticus by the fact that in the latter there is only one contraction of the heart for every two beats of the pulse, while in pulsus bigeminus there are two contractions of the heart, which follow one another rapidly and are separated from the preceding and succeeding contractions by a longer pause. For every two beats of the pulsus dicroticus there occur, as in the normal pulse, only two heart tones, while in pulsus bigeminus four heart tones are audible. An accurate reproduction of the pulsus bigeminus is found in Table 9 under C in the first volume of my "Contributions." This was an instance of an animal that had been curarized and then poisoned with potassium cyanide, and shortly after being poisoned, the vagi in the neck were sectioned. We observed the pulsus bigeminus appearing soon following section of the second vagus, after the pressure and pulse rate were increased in consequence of the operation.

The first time that I observed this phenomenon was when I permitted the longer suspension of artificial respiration in curarized animals, when beginning failure of the left ventricle occurred. One soon sees a marked excitation of the inhibitory spinal nervous system after the onset of suspension under the rising tension in the aorta, which is evidenced by a considerable diminution of the frequency of the pulse and an increase

in the pulse volume; later, under reduction of the arterial pressure, excitation of the inhibitory spinal nervous system produces paralysis in the same place; the pulse frequency accelerates, and then under further reduction of arterial tension the pulsus bigeminus appears; it first follows each pulsus tardi which manifests overwhelming fatigue of the left ventricle preceding its complete standstill.

Through greatly prolonged suspension of artificial respiration, which was undertaken in the animals with sectioned vagi, and where the heart had been deprived of the influence of the inhibitory spinal nervous system, we see the pulsus bigeminus appear under circumstances similar to those in which the vagi are intact, that is, after the arterial tension is appreciably reduced and shortly before considerable reduction in the frequency of the pulse takes place, the low pulsus tardi appears, which portends the failure of the left ventricle.

Finally, I discovered that this phenomenon would appear every time, shortly after poisoning in an animal with the vagi sectioned, when a substance was administered which stimulated the heart through the entire inhibitory spinal nervous system.

I concluded from these facts, that two conditions are necessary for the appearance of the pulsus bigeminus.

(1) The heart must be released from the influence of the inhibitory spinal nervous system, and also

(2) There must be some agent circulating in the blood, which increases the irritability of the cardiac component of the inhibitory spinal nervous system, which is still functional.

If this conclusion is correct, we can obviously conclude from the appearance of the pulsus bigeminus in patients that a paralysis of the inhibitory spinal nervous system exists, and the prognosis under such circumstances would be unfavorable.

Up to the present time, I have been enabled in three or four cases to demonstrate the pulsus bigeminus in man; in two of these death ensued shortly after its appearance.

The following case, which came under my observation toward the close of last year, demonstrates a variation of the pulsus bigeminus; I designate it with the name of "pulsus alternans." It has certain features in common with the pulsus bigeminus in that the normal rhythm is not replaced by an arrhythmia but by a new extraordinary rhythm in which two consecutive pulses are in closer approximation to one another: it involves a succession of high and low pulses, in such a manner that a low pulse regularly follows a high pulse and this low pulse is separated from the ensuing high pulse by a shorter pause than that between it and the preceding high pulse. The following curve taken by me with the aid of Marey's sphygmograph from the radial artery of a patient, which forms

the starting point of this communication, gives us a more precise idea of this type of pulse.

I present the case history in detail, since it appears more interesting in its various aspects.

Observation

H. W., laborer, forty-seven years old, entered my service on October 16, 1871, had for years engaged in heavy labor and was acknowledged an intemperate person. He had smallpox a long time ago and recovered from pneumonia seven years ago. At Christmas time 1870, he held a heavy barrel of spirits against his chest, which, together with other laborers, he attempted to carry up a stairway. He felt no distress at the time, but in a short while he became short of breath, which so rapidly increased in intensity that he was obliged to discontinue the work he was engaged in, but also consumed fully two hours to cover the short distance to his home. After the patient had remained in bed for eight days, he attempted to resume his occupation, which precipitated an exacerbation of the dyspnea which soon again returned him to the sick-bed; however, he suffered from neither cough nor pains in the chest. After a month he felt considerably improved so that he again believed himself able to resume work, which he now pretended could be carried out without discomfort. This condition of subjective well-being lasted until June, 1871. At this time, in consequence to more strenuous effort, great air-hunger occurred, but again without cough or pain in the chest. Renewed forbearance of activities brought no relief this time. Soon after this he began to complain of a disagreeable feeling in the epigastric region. He was thereby moved to seek aid in Charity Hospital.

On admission on October 16 the following was noted:

Patient complains of air-hunger and pains in the epigastrium; in addition, marked objective dyspnea is present; cough is absent.

> Temperature, 37.8
> Pulse frequency, 120
> Respiratory frequency, 40.

Orders: One spoonful of castor oil, meanwhile a mustard plaster, and Sol. Potas. acetate, 10.0 in 200.0; 1 teaspoonful every two hours.

A more thorough examination on June 18 revealed the following:

The patient has a fairly robust constitution, a fairly generous panniculus, good musculature. The dyspnea has become definitely decreased. The thorax is well developed, although the excursions are limited. There

is no cough. Below the scapulae are occasional rales. The cardiac dulness is increased in all directions and abnormally intensified. The apex beat is distinctly visible and palpable in the sixth intercostal space, is one inch in breadth and lies beyond the mammary line. Auscultation in the region of the apex beat reveals a loud systolic murmur, and a low diastolic sound, in addition to an otherwise normal tone, which accentuates the diastolic pulmonic arterial tone. The radial artery was narrowed, from abnormal tension; low pulse. The jugular veins distended, the liver greatly enlarged. Urine scant; specific gravity 1.015.

The temperature also remained normal during the ensuing days, while the frequency of the pulse varied between 116 and 124; the urine continued to be scant and red.

On October 19, the infusion of digitalis was ordered, 1.0 to 200.0; 1 teaspoonful every two hours

On the twenty-second, after the use of 2 Gms. of digitalis, the frequency of the pulse dropped to 104, the respiration to 24, the 24 hour urinary output rose to 2,000 c.c., the specific gravity remained the same, 1.014; the temperature 37.1.

Order: Digitalis discontinued; instead, the patient is to be given a solution of potassium acetate 3.0 to 150.0, one teaspoonful every 2 hours.

I personally first carefully examined the circulatory apparatus on the thirtieth of October. This examination disclosed the following:

The apex beat in the sixth intercostal space, near but beyond the mammary line, the area 1 inch in breadth; a second, but weaker systolic elevation is found, situated more to the inner side, in the fifth intercostal space. Beyond the apex beat, observed at a distance from its abnormal location, there also appears an abnormal pulsation, consisting of a diffuse systolic elevation of the entire precordial region, which also involves the lower portion of the sternum and is most marked in the region of the costo-cartilaginous articulations. One observes a systolic retraction at the point of the epigastrium, and another in the fifth and sixth intercostal spaces near the previously described elevation, in the neighborhood of the apex of the heart. Auscultation in the region of the apex-thrust reveals a moderately loud systolic murmur, and a fairly loud diastolic tone; over the xiphoid process, two loud tones, and in the vicinity of the pulmonic area, a systolic murmur and a definitely accentuated diastolic tone. Radial arteries quite broad; abnormally tense. When one palpates the artery more firmly, distinct regular alternations in the height and depth of the pulse are noted.

> Pulse frequency, 96
> Respiration, 20
> Temperature, 37.3.

Urine volume 2,000 c.c., specific gravity 1.014.

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I made the diagnosis of hypertrophy and dilatation of both ventricles associated with sclerosis of the aorta: the pulsus alternans was the same as on preceding days.

. . . .

Under the influence of an increasing icterus, which finally reached a profound degree, an increasing accumulation of abdominal fluid, persistent restlessness and delirium, marked collapse finally ensued. Death occurred on December twenty-seventh.

. . . .

The post-mortem examination conducted twenty-four hours later (Dr. Wegener) revealed the following:

There was marked edema of the lower extremities, considerable ascites. The skin was of an olive color, and the visible mucous membranes intensely icteric. There was an area of hemorrhagic infiltration in the conjunctiva of the right eye. The ascitic fluid was of a yellowish-brown color.

The diaphragm on the right side reached the lower edge of the fifth, and on the left the upper edge of the seventh rib; in the left thoracic cavity was a copious serous, icteric-colored exudate, also considerable fluid in the pericardium.

The heart was greatly enlarged in all dimensions, its musculature quite firm; it contained a great quantity of fluid blood intermingled with clots. On the anterior wall of both ventricles were large fibrous patches, both ventricles were greatly dilated and hypertrophied. On the inner surface of the left ventricle, closely adherent to the trabeculations, was a thick continuously adherent, laminated, cystic and softened thrombus, of an icteric color, which in part was a dirty brown and in its inner portion of a greenish-yellow color. The valves, with the exception of having a pronounced icteric color, were normal. The ascending limb of the aorta immediately above the valves was dilated, in the upper portion of the arch, a prominent parietal thrombus was firmly adherent to the intima.

. . . .

Epicritical Remarks

I. In this detailed case history, it is possible without difficulty to distinguish five periods of time.

In the first period the illness had its inception, that is to say, its latent course, at which time the hypertrophy of the left ventricle was so well compensated that the patient experienced no appreciable discomfort and his condition could have been recognized only fortuitously by even a meticulous and expert observer.

The second period begins with the catastrophe; the patient, together with other laborers, carried a heavy burden held against his chest, up a stairway. This period is characterized by the fact that the patient became annoyed by air-hunger, at first only under strenuous exertion, later at rest, which was accompanied by a disagreeable discomfort in the epigastrium. As the patient, on this account, sought admission to the Charity Hospital, one interpreted the signs, namely, a pulse of high frequency, that ranged between 116 and 120, marked narrowing of the radial arteries, weak pulse, distention of the jugular veins, marked enlargement of the liver and scanty red urine of high specific gravity, but without a trace of hydrops, as occurring with the appearance of dilatation and hypertrophy of both ventricles. One was obliged to conclude, that it resulted from a loss of the compensatory mechanism of the left ventricle, leading to an engorgement of the pulmonary circulation, that as a result of this engorgement, hypertrophy of the right ventricle occurred, and finally, as the increased activity of the right ventricle began to fail, an engorgement of the systemic veins occurred.

During the third period, which had its inception shortly after the onset of the illness, and when I could express myself, the patient's condition was generally improved by digitalis. He walked about the room, participated in the care of other patients, complained only of short periods of palpitation; the presence of disturbance of compensation was evident only by the enlargement of the liver and the distention of the jugular veins. In this period, which reached from October twenty-second to November seventeenth, we came upon a characteristic pulsus alternans.

The fourth period began when the urinary volume again became diminished. Then predominantly digestive disturbances occurred: the patient complained of nausea, anorexia and diarrhea. Later, the respiratory apparatus became involved: attacks of air-hunger appeared, persistent cough with slimy, reddish colored expectoration, that ultimately became a dark reddish-brown, revealed the presence of hemorrhagic in farcts; now for the first time edema of the lower extremities appeared. At this stage, the definite, unmistakable pulsus alternans that earlier was present in the carotids disappeared.

The fifth stage extended from the thirteenth to the twenty-seventh of December (the latter being the day of death), and was primarily characterized by intense irritation of the entire digestive tract: the diarrhea became more marked; added to this was violent recurrent vomiting, also icterus, and then the enlarged liver began to diminish in size. During the rapid diminution in the volume of the liver the patient developed an agitated delirium, which simulated an acute mania, during which time the icterus increased in intensity, and extravasations appeared in the conjunctiva and in the skin of the abdomen. Death occurred during the delirium and following severe collapse.

According to the accustomed view, all the complaints could be attributed to arteriosclerosis, as revealed by the post-mortem. In this observation, it cannot be denied, that at least in a series of cases, the sclerosis occasioned conditions which hampered considerably the emptying of the left ventricle. It will thus be the case when as the result of degenerative dilatation of the aorta the elasticity of the walls of its greater arterial tributaries becomes diminished. Furthermore, these vessels assume the role of rigid tubes, and the force must be conceivably greater than ordinary, in order to deliver a specified volume of blood to a given place. But in the great majority of cases such a change in the large arteries does not occur; notwithstanding considerable hypertrophy and dilatation of the left ventricle, and without other significant findings in the body which could be attributed to the disease of the heart, we find only slight or moderate degrees of sclerotic prominences in the lining of the aorta, which could not appreciably affect the elasticity of this vessel. Finally, one encounters cases, which in their clinical course, completely coincide with these, and in which obviously identical etiologic factors were at work; the post-mortem, with the exception of dilatation and hypertrophy of the left ventricle, revealed no changes in the large arteries.

One observes from this, that arteriosclerosis could not possibly be the direct cause of the affection of the left ventricle, as is generally believed. It was Senhouse-Kirkes,* who first called my attention to the premise, that arteriosclerosis is the result of protracted high-grade tension in the aorta. Therefore, I believe, the first clue to a correct insight of the affection, is not only an understanding of the manner of its production, but also of its pathologic significance. If I did not completely accept the opinion of the authors mentioned in the foregoing discussion, and thus doubt them, that the abnormal tension in the aorta directly favors the development of arteriosclerosis, then I may also assert, that arterial disease as a rule is not the cause of the heart affection, but rather more often that both conditions are common co-effects.

Arteriosclerosis and hypertrophy of the left ventricle, especially in the aforementioned older persons, occurs particularly, according to more recent English experience, when the abuse of alcohol is combined with muscular exertion. If we examined such individuals more closely, the signs of abnormal tension in the aorta are soon evident. And the latter are apparent in those individuals in whom no degeneration of the aorta or its tributaries is disclosed by post-mortem. The basis for hypertrophy of the heart under these circumstances is readily seen; it can be none other than the increased arterial tension. Since the left ventricle empties its content into an arterial system of such great tension, that is, since the ventricle must raise the blood mass to a greater height than under normal

*See page 472.—F. A. W., 1940.

conditions, its bulk must increase, like all muscles, so that it is forced to perform greater work in order to effect normal conditions of nourishment. According to Senhouse-Kirkes, as we have seen, arteriosclerosis here has the same cause as hypertrophy of the left ventricle, but I have recorded on an earlier occasion (No. 29 in the *Wochenschrift* of the previous year), that in cases with which I dealt, yet another condition must be considered than abnormal tension of the arterial distribution; this is the slowing of the blood stream. Inasmuch as alcohol probably increases the tone of the arterial musculature, and thus lessens the outflow from the aorta, it not only increases the tension in the arteries but also diminishes the rapidity of the arterial blood stream. I have expressed myself in such detail in the last consideration on how the origin of the sclerosis may be favored, that I abstain from further explanations here regarding this belief, which in all events is evident; also, according to my concept, hypertrophy of the heart and arteriosclerosis are common basic conditions: from which on the one hand the increased tension in the aorta results and consequently, hypertrophy of the left ventricle; while on the other hand, slowing of the blood stream in the larger arteries occurs, and consequently, upon this the sclerosis is dependent.

Cases of this type, regardless of pulsus alternans, are more frequently observed in the hospitals but also are not infrequently encountered in private practice among the middle classes. Here, as a rule, excessive muscular exertion plays no noteworthy role, and likewise, the abuse of alcohol is not of constant etiologic importance. According to my experience, two other influences prevail: excessive smoking of tobacco and congestion of the portal system, which become aggravated by sedentary living and over-eating. I believe it is possible to approach these points by other means.

Strange and therefore unbelievable as it appears in our case, the illness first began as the patient rolled the heavy keg of spirits against his chest. As the post-mortem showed, there were no traces of partially healed severe acute disease of the respiratory or circulatory apparatus in the body, and herein, the complaints of the patient harmonize, since he complained only of dyspnea which appeared soon after the catastrophe, but without cough or pain in the chest. From the standpoint of fever, as we see, there was likewise nothing to mention. It required but slight reflection to understand, that the subtle events which we noticed did not indicate signs of disease, but indicated disturbance in compensation, in other words: at the time of the catastrophe, while the patient was under the influence of excessive and frequent use of alcohol and repeated and unaccustomed muscular exertion, hypertrophy of the left ventricle, as well as the arteriosclerosis, had already been present for a considerable time. The patient showed no signs of trouble, while the disturbances in the aorta, resulting from his faulty manner of living, remained fully compensated. Through the unaccustomed great muscular exertion that

II. According to the accepted view, one of the first signs of beginning congestion in the venous system of the body in organic disease of the circulatory and respiratory apparatus, is a hydropic swelling of the lower extremities. I also held this view for a long time. The first case that puzzled me, I observed for nearly ten years, was a young colleague, who consulted me owing to a persistent hepatomegaly. By careful examination I found a very extensive, uniform enlargement of the liver, but also insufficiency of the aortic valves. The anamnesis gave no clue, and made independent and more profound disease of the liver probable; it overshadowed the probability of a swelling from congestion of the hepatic venous system. But why did hydrops fail to appear?

After my attention was once focused in this direction, other similar cases soon came under my observation, and I gradually became convinced, that no hydrops, but swelling of the liver, was the first sign of beginning congestion of blood in the venous system of the body. Our case in comparison offers no exception to the rule, it is contrarily to be viewed as an example of usual occurrence. The old rule in regard to swelling of the liver finds new significance, in that the heart must always be considered and examined, before the diagnosis of liver disease is definitely made. In retrospect, one can definitely determine the nature of this swelling in those individuals who show no trace of hydrops, when they present uniform enlargement of the liver with smooth surfaces; but one must first undertake a careful examination of the heart.

III. Owing to my absence at the autopsy, microscopic examination of the liver was neglected. I greatly regret this, as the course of this case in the final period had aroused conjecture that the patient had succumbed to acute atrophy of the liver. For the acceptance of this view, we find the following evidence: (a) the rapid diminution in the size of the liver as determined by palpation; (b) the ever-increasing icterus; (c) the peculiar form of delirium; and (d) the absence of febrile reaction. Were my conjecture correct, the case would offer a new contribution to the accepted teaching, that the acute degeneration of the parenchyma of the liver and the resulting rapid diminution in size of the liver, were accidents in the course of the various acute as well as chronic, severe as well as mild, diseases of the liver, and we would presume, that the diseases with icterus are included in this group. I recollect, moreover, of having observed various cases of heart disease, in which death occurred with phenomena similar to those of the foregoing case.

IV. In conclusion, just a few words regarding our observations on pulsus alternans. A review of this case from the beginning of the epicrisis must strengthen the conviction that this phenomenon was influenced, at least in part, by the digitalis. I observed this phenomenon during the third period of the illness; that is, in the interval during which the patient improved while using greater quantities of digitalis, when existing failure of compensation could be recognized only by enlargement of the liver and distention of the jugular veins. With the diminution in the action of the digitalis, the pulsus alternans began to disappear, and finally was only distinctly seen in the carotids. But digitalis belongs, as I have shown, to those agents, which stimulate the inhibitory nervous system of the heart. Following this complete consideration, we are justified in concluding the discussion of the close relationship of pulsus alternans and bigeminus.
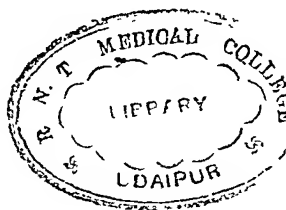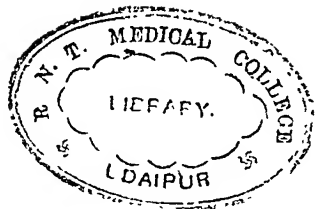
1876

WILLIAM RICHARD GOWERS

DESCRIPTION OF THE RETINAL VESSELS
IN HYPERTENSION

SIR WILLIAM RICHARD GOWERS

(Courtesy Journal of Nervous and Mental Disease
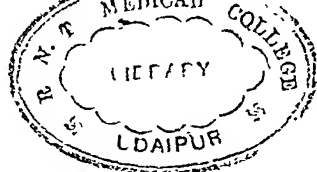
SIR WILLIAM RICHARD GOWERS

(1845-1915)

ON MARCH 20, 1845, William Richard Gowers was born in London. He received his academic training at Christ Church College School, Oxford. When he was sixteen years of age, Gowers was apprenticed to a country surgeon at Coggeshall, Essex. He received his medical education at University College, London, and in 1867 qualified for membership in the Royal College of Surgeons. Two years later he received the degree of Bachelor of Medicine from the University of London. In 1870 he received the degree of Doctor of Medicine and was awarded the gold medal for his high standing in the class.

After graduation, Gowers was appointed medical registrar to the National Hospital for the Paralyzed and Epileptic. He also continued his position as private secretary to Sir William Jenner, a position which he had occupied during his student days. Gowers deeply appreciated his contact with Sir William Jenner, and Jenner helped Gowers in his brilliant career.

In 1872, Gowers was appointed assistant physician to University College Hospital and became physician to that institution in 1883. Meanwhile, in 1873, he was appointed to the rank of assistant physician at the National Hospital for the Paralyzed and Epileptic. He became a physician there in 1883. On his retirement in 1888, he was appointed consultant. He also served for many years at the University Medical School as a teaching assistant and later was appointed professor of clinical medicine.

Gowers was greatly interested in the diagnostic value of the ophthalmoscope and, in 1876, he published an important paper in the "British Medical Journal" entitled: "The State of the Arteries in Bright's Disease." Because this article contains his classic description of the retinal vessels in the presence of hypertension, we are including it in our CARDIAC CLASSICS. His more detailed work, "A Manual and Atlas of Medical Ophthalmoscopy," was published in 1879. Although the importance of the ophthalmoscope was realized by others before Gowers, this publication, which was thorough and systematic, brought ophthalmoscopy into a much wider use in general medicine than had been the case before. A fourth edition of this deservedly successful book was published in 1904.

In 1879, Gowers was elected a fellow of the Royal College of Physicians. He had qualified for membership in that organization in 1875. In 1880, he gave the Goulstonian Lecture, choosing for his subject, "Epilepsy and Other Chronic Convulsive Diseases." This address was published in 1881.

Gowers, at the beginning of his career, showed a special interest in the diseases of the nervous system. In 1876 he published an essay: "On 'Athetosis' and Post-hemiplegic Disorders of Movement." In 1877 he wrote on "The Diagnosis and Treatment of Auditory Nerve Vertigo," and in 1878 he published an important paper on chorea, "On Some Points in the Clinical History of Chorea."

His interest in neuro-anatomy led Gowers to the discovery of the tract of fibers in the gray matter in the ventral and lateral funiculus of the spinal cord: the fasciculus arteriolateralis superficialis, also known as Gowers' tract. His discovery was first published in 1879 in his lecture, "The Diagnosis of Diseases of the Spinal Cord." An enlargement of this lecture as a book was published in 1880, under the same title.

[603]

Gowers is also remembered for his method of estimating the percentage of hemoglobin and the number of corpuscles in the blood. In December, 1878, he presented the details of his hemoglobinometer before the meeting of the Clinical Society of London. His method was used until 1901, when Haldane's modification took its place.

In 1885, Gowers published his famous work: "Lectures on the Diagnosis of Diseases of the Brain," in which he correlated the observations of Hughlings Jackson (1834-1911), Paul Emil Flechsig (1847-1929), Eduard Hitzig (1838-1907) and David Ferrier. Between 1886 and 1888 this unceasing and seemingly untiring worker published his two volumes, "A Manual of Diseases of the Nervous System." In 1899, the third edition of the work was published.

Gowers published another monograph in 1892; it consisted of his Lettsomian Lectures on "Syphilis of the Nervous System." He had delivered these lectures in 1890. Many years previously, in 1879, he had delivered his first Lettsomian Lecture on syphilis of the nervous system. It was Gowers' belief that, apart from embolism and injury, sudden hemiplegia occurring between the ages of twenty-five and forty-five years is very seldom the result of anything other than syphilis.

In 1897, Gowers, on the occasion of the Diamond Jubilee of Queen Victoria, received the honor of knighthood. Meanwhile, his health, which had become severely strained by the labor of publishing so many exhaustive volumes and the worries of a large practice, became dangerously poor. To improve his health he took a long voyage and vacation to South Africa. He returned to England feeling better. He maintained his practice and continued to make contributions to the medical literature. In 1907 he published his last important work entitled: "The Border-Land of Epilepsy; Faints, Vagal Attacks, Vertigo, Migraine, Sleep Symptoms, and Their Treatment."

Sir William was the recipient of many honors. He was a fellow of the Royal Society of London. He received the honorary degree of Doctor of Medicine from the University of Dublin and that of Doctor of Laws from the University of Edinburgh. He was elected an honorary fellow of the Royal College of Physicians of Ireland. He was also an honorary member of the following societies: the American Neurological Association, the Netherlands Society of Psychiatry and Neurology, the Russian Society of Medicine, the Royal Society of Science of Upsala, and the Society of International Medicine of Vienna.

Sir William married the daughter of Frederick Baines of Leeds. She died in 1913, leaving four children: two sons and two daughters.

Sir William Gowers died on May 4, 1915, after a long illness.

THE STATE OF THE ARTERIES IN BRIGHT'S DISEASE[*]

By

W. R. GOWERS, M.D. Lond.

*Assistant-Physician and Assistant-Teacher of Clinical Medicine, University College
Hospital*

THE OPHTHALMOSCOPE is of service to the physician, among other uses, because it enables him to see the termination of a minute artery and vein, and to gain direct evidence of their condition, such as is to be obtained in no other way. When retina is free from local disease, there is no reason to believe that the retinal artery and vein differ in their condition from other arteries and veins of the same size, and, therefore, any marked change in their state, apart from cerebral or ocular disease, may be taken as evidence of a similar change throughout the vascular system.

The object of the following paper is to bring forward certain facts concerning the retinal vessels in Bright's disease, and I believe the facts warrant this conclusion; that, in chronic Bright's disease, the arteries of the retina are sometimes of normal size and sometimes very distinctly lessened in size; that this diminution in size depends upon contraction; and that this visible contraction stands, as a rule, in direct proportion to the tension of the arterial blood, as measured by the incompressibility of the radial pulse.

A few words are necessary as to the manner in which the size of the arteries is estimated. Their condition can only be seen under considerable magnifying power. Examination by the indirect method does not, as a rule, give sufficient enlargement unless a lens of very long focus be employed, as in Carter's demonstrating ophthalmoscope. Now and then, if the eye be hypermetropic and the vessels very distinct, the indirect examination with a lens of three- or four-inch focus will suffice; but as a rule, it is necessary to employ the direct method of examination. If the pupil be small, it must be dilated with atropine, since it is often necessary to trace the vessels for some distance from the disc.

There is unfortunately no method of applying any gauge to the vessels; their size must be estimated by the eye. The change in size may be judged of absolutely or by comparison with the veins. For an absolute estimate of their size, familiarity with their normal appearance under direct examination is, of course, necessary. Further, as the degree of

magnification varies with the refractive power of the eyeball, this must be allowed for. It may generally be estimated by noticing the apparent size of the disc.

The change in the size of the arteries is frequently such as to be recognised at once; there is no need for comparison with the veins. The reduction in size may be so considerable, that even the primary branches of the central artery are so small that their double contour is recognised with difficulty, and it may be unrecognisable even by direct examination, the arteries being, as in one example I have to show, visible only as lines.

In other cases where the diminution in size is slighter, it can be most conveniently estimated by comparing the arteries with the veins. The distribution of the arteries and veins corresponds approximately, not exactly. Sometimes two arteries accompany one vein, sometimes one vein corresponds with two arteries. But in each eye there are usually some single branches of arteries and veins which have an identical course and distribution, run side by side, and are available for comparison. When this is the case, it will be found that, as a rule, the width of the artery is about two-thirds or three-quarters that of the vein. When the artery bears less proportion to the vein than this, it is usually due to one of three causes: 1. General venous distension, as in cyanosis; 2. Impediment at the sclerotic ring, by which the entrance of blood into the arteries is impeded, and its exit from the veins is also hindered, in which case the arteries are narrow and the veins distended as in certain stages of optic neuritis; 3. Contraction of the arteries. In the two former cases, the veins are, of course, abnormally large, and their abnormal size is generally easy of recognition. In the latter case, the veins may be normal in size or may be smaller than natural. If they be smaller, the diminished proportionate size of the arteries is of still greater significance. It is necessary, therefore, to be familiar with the normal size of the veins, in order to estimate the size of the arteries by comparison. From their darker colour, their size is easily noted, and the size of the arteries is readily estimated by comparison.

The arteries may be of normal size upon the optic disc, and yet present very marked reduction in size on the retina, a little distance from the disc. An artery may leave the disc beside a vein to which it bears its normal proportion, and, after a little course, without giving off any visible branch, may diminish to one-half or one-third of the size of its accompanying vein.

From what has been said, it will be obvious that these changes in the relative size of the vessels possess most significance when the retina has not undergone the special changes to which it is liable in chronic Bright's disease. Exudation within the sclerotic ring, compressing the vessels, alters their relative and absolute dimension, as I have stated. This is well seen in ordinary optic neuritis. I believe that it is rare in Bright's dis-

ease for the neuritic change to be sufficient to produce this effect. Certainly, in cases of albuminuric retinitis, the actual contraction may often be recognised as something quite out of proportion to the retinal change.

As I have said, the rule that, when the arterial tension is increased, the retinal arteries may be seen to be contracted, is general, but not universal. This is in accordance with what might be expected from the various conditions which are known to influence, on the one hand, blood-tension, and, on the other, arterial contraction. Moreover, local influences may cause local modifications. The most notable exceptions to the rule, which I have met with, have been in cases of local retinal disease.
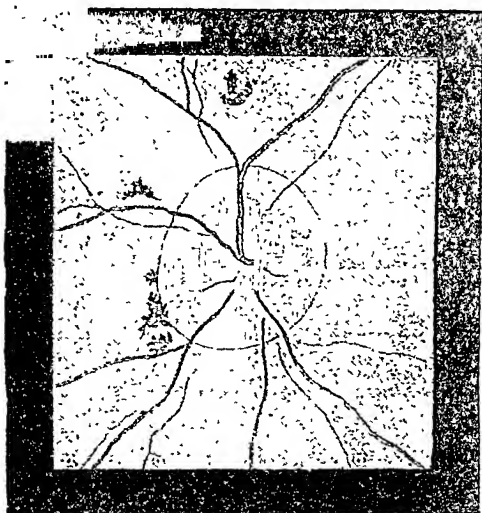
Fig. 1.

The following are some of the facts on which the conclusions described have been based. The incompressibility of the radial pulse was employed as the estimate of arterial tension. When practicable, my own estimate has been corroborated by a sphygmographic tracing, or by an independent opinion.

On opposite sides of a ward in University College Hospital, there recently lay two patients (under the care of Dr. Reynolds), whose cases illustrated in the most marked manner the relation of blood-tension and the state of the retinal arteries. The one case was that of a man, fifty-eight years of age, whose illness had commenced gradually, with shortness of breath and weakness, two years before. Slight oedema of the legs had existed for only one month before his admission. His urine contained one-third of albumen; had a specific gravity of from 1 005 to 1.008,

and contained numerous casts, granular and hyaline, with some degenerated epithelium, both free and within the casts. His retinae were normal in appearance; the arteries of full size, presenting not the slightest evidence of contraction. His pulse was full, but very soft and compressible. There was no evidence of cardiac change. The other case was that of a man, aged 46, whose symptoms resembled those of the first. They began with shortness of breath and swelling of the legs nine months before. His urine had a specific gravity of 1.007 to 1.010, and contained from one-third to one-half albumen; its quantity was from two to four pints, and it contained many casts, granular, hyaline, and epithelial. His retinae presented evidence of slight disease. The optic discs (Fig. 1) had softened outlines, and their surface was reddish-grey, paler in the vicinity of the vessels. There was little, if any swelling. The veins were smaller than normal; in the left eye, one only approached the average size. The arteries presented a greater reduction in size than in any case I have seen. Even on direct examination, they were visible only as lines, no double contour being recognisable, although they were quite distinct. A few minute white dots existed in each eye near the macula lutea, and in each there were a few small extravasations. The vessels were similar in the two eyes. The disc in the right was a little less grey than
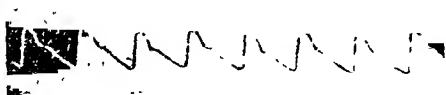
Fig. 2.

in the left. Vision: R. = one-sixth; L., one-twentieth. There was no peripheral limitation of the fields of vision. The pulse was extremely hard; the artery felt like a whipcord under the finger, and was almost absolutely incompressible. The strongest pressure which could be put upon it with a Marey's sphygmograph did not modify its character. The accompanying tracing (Fig. 2) was taken under the highest available pressure (about four hundred *grammes*).

The sepia drawing now passed round represents the fundus oculi in a case of chronic Bright's disease, the sequel of an acute attack 12 years previously, the patient having in the meantime had at least two other acute attacks. The urine was loaded with albumen and contained granular and fatty casts. The retina presents abundant soft-edged white areas and also many striated extravasations; most of these had appeared during the preceding ten days. The disc is concealed by oedema. The veins are of normal size; the artery at the papilla is rather smaller than natural, its branches being not more than half the size of the veins; but, a little distance beyond the limits of the papilla, they disappear. The veins can be followed distinctly on the retina, but the arteries can only be seen as

dim lines here and there. It may be suggested that this appearance is due only to the opacity of the retina; but the fact that they can be dimly seen here and there as lines suggests that their indistinctness is due in part only to the opacity of the retina, in part also to their reduction in size. The greater extent of this reduction on the retina than on the disc suggests that it is not due to their obstruction at the lamina cribrosa, but to their active contraction. The facts of other cases give support, I think, to this view. This patient's pulse was also very hard and incompressible. He insisted on going out of the hospital, but died comatose a few days afterwards. A post mortem examination was obtained by Dr. Burton, one of the resident assistants at the hospital. The kidneys were found lessened in bulk, increased in consistence, and moderately granular on the surface. The heart was hypertrophied.
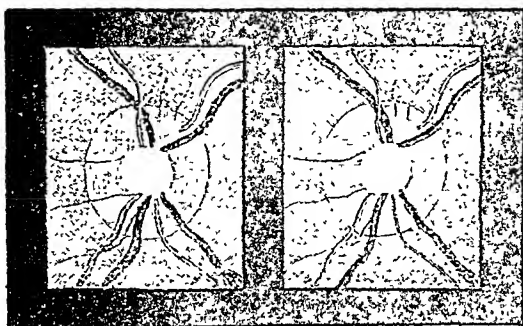
Figs. 3 and 4.

The next two sketches (Figs. 3 and 4) represent the optic disc of a patient (under the care of Dr. Ringer) suffering from acute Bright's disease passing into a chronic state. The first was made six weeks after the onset. The retina presented a few small haemorrhages, white dots around the macula lutea, with a few larger white areas. The arteries and veins were normal in size, the former being just two-thirds the diameter of the latter. The pulse was soft and compressible, giving no evidence of increased blood-tension. When the second sketch was made, six weeks later, the patient's general condition had improved, the albumen in the urine was less, but the casts had become fatty; the retinal changes had become considerably less; the white areas had lessened. The retinal arteries, however, presented distinct diminution in size compared with their previous condition. The veins were apparently of the same size as when the former sketch was made, while the arteries had diminished to one-half the size of the veins. The pulse also presented a marked alteration. It had become distinctly harder and less compressible. The change seemed to have taken place only a short time before the sketch

was made; for, on my calling the attention of the resident assistant to its character, it struck him at once as quite different from that which had been its character a short time before.

I have repeatedly formed, from an inspection of the retinal vessels, an opinion as to the arterial tension, which, I afterwards found, on examining the pulse, was correct. In order, however, to obtain some evidence which might be without even unconscious bias, I asked my friend Dr. Coupland to be good enough to examine the pulse in a series of cases of Bright's disease whilst I examined the retinal vessels. Each wrote down independently the result of the examination. Five cases were examined, and the results agreed in four. In one case, they differed; but in this the fact that there were many retinal haemorrhages may, as I have already said, explain the absence of arterial contraction, although the pulse was hard. The following are the details of these examinations.

CASE I.—Arteries small, about one-half the diameter of the veins; moderate contraction (retina healthy, except for a few haemorrhages; disc clear); pulse moderately tense, *i.e.*, moderately incompressible.

CASE II.—Arteries one-half the size of the veins; moderate contraction (retina perfectly normal); pulse incompressible (about as the first case).

CASE III.—Arteries nearly two-thirds the size of the veins; very little contraction (many extensive retinal haemorrhages); pulse markedly incompressible.

CASE IV.—Arteries less than half the size of the veins; great contraction (retina normal, disc clear); pulse very incompressible.

CASE V.—No diminution in the size of the arteries; retina healthy; pulse soft and compressible.

Thus, in the case in which the retinal arteries were smallest, the arterial tension was greatest; that in which the arteries and the retina were normal presented no excess of arterial tension; the two others, in which there was a moderate contraction of the arteries, presented a moderate increase in the arterial tension.

There is, of course, nothing new in the fact that the retinal arteries are small in Bright's disease; it has long been remarked as a common feature in albuminuric retinitis, and is shown plainly in the best illustrations of this change (as in those of Liebreich). But it is usually regarded as a consequence of the retinal change, and the points on which I would insist are that it occurs also quite independently of the retinal change, and stands commonly in direct relation to another condition—the blood-tension.

It is hardly necessary for me to point out the bearing of this conclusion on the theory of Dr. George Johnson, which ascribes the increased tension of the blood in Bright's disease, in part at least, to contraction of

the minute arteries. It constitutes, I think, a direct proof of the correctness of the theory, which has hitherto derived its chief support from indirect inference from pathological facts. If the tension of the arterial blood and the arterial contraction occur in common proportion, they must stand in a causal relation to one another. But the blood-tension cannot be the cause of the arterial contraction, because it is well known from physiological experiments that the tendency of increased blood-tension is, through the depressor nerve, to cause relaxation of the arterioles. But, on the other hand, as the immediate effect of contraction of the arterioles must be an increase in the arterial blood-pressure, it is reasonable to conclude that such is the sequence of events in the phenomena under consideration; that, although the two phenomena may be in part the result of a common cause (altered state of the blood), the contraction of the arteries, seen in those of the retina and inferred to exist elsewhere, is, in part at least, the cause of the increased blood-tension.

The practical use of inspection of the retinal vessels is perhaps less than its pathological value, but it is, I think, considerable. It is true we can generally ascertain the amount of arterial tension more readily and more surely by feeling the pulse than by looking at the retinal vessels. But sometimes the incompressibility of the pulse cannot readily be estimated, on account of its smallness and the amount of subcutaneous fat or oedema. In these cases, retinal inspection may be useful. Moreover, as affording definite information regarding the pathological processes in different cases of Bright's disease, it will, I think, have considerable value; and some facts which have come under my observation, at present too few and isolated for more than mention, make me hope that ultimately it may help us better to distinguish between morbid states included under the term and at present imperfectly distinguished.

1877

JULIUS FRIEDRICH COHNHEIM

DESCRIPTION OF PARADOXICAL EMBOLISM

Dr. Jul. Cohnheim, ord. Prof.

JULIUS FRIEDRICH COHNHEIM
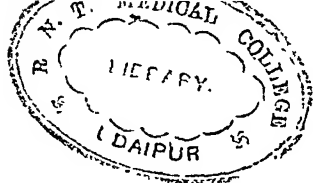
JULIUS FRIEDRICH COHNHEIM

(1839-1884)

"Without blood vessels, no inflammation is possible."

—Cohnheim.

*J*ULIUS FRIEDRICH COHNHEIM was born on July 20, 1839, in the town of Demmin, Pomerania, a province in Northern Prussia. He received his gymnasium training at Prenzlau, and in 1856 began the study of medicine at the University of Berlin. Later he studied at the University of Würzburg. Cohnheim pursued his studies in an ardent manner, and under the influence of Albert von Kölliker, developed into an expert histologist. He remained in Würzburg until the spring of 1860. He then studied for a few months at Greifswald in Pomerania and returned to the University of Berlin.

At Berlin he passed his examination for the degree of Doctor of Medicine. He then prepared his thesis under the direction of Rudolf Virchow (1821-1902), the title of his dissertation being "De pyogenesi in tunicis serosis." While working under Virchow at the Pathological Institute of the Charity Hospital in Berlin, Cohnheim became acquainted with Friedrich von Recklinghausen (1833-1910) and Edwin Klebs (1834-1913). The inspiration he received from these three men no doubt helped him in his decision to devote his life to science.

Meanwhile, Cohnheim's father, who had been forced by circumstances to leave Germany for Australia, returned home suffering from a fatal illness and in July, 1862, died. It became necessary for the son to contribute to the support of the family, but he spent what spare time he could in the study of normal and pathologic histology and in learning the methods of chemical investigation.

In 1862, Cohnheim came under the influence of Ludwig Traube (1818-1876). He became one of Traube's students and gained not only clinical experience from this great teacher but also a knowledge of experimental physiology. In appreciation, Cohnheim later dedicated his "Lectures" to Traube's memory. That same year, 1862, Cohnheim passed the state examination in medicine.

Cohnheim served as a surgeon in the Prussian Army in the German-Danish War of 1864. On the death of his brother, Albert, a victim of the war, Cohnheim, being the sole support of his mother, was released from military duty. He then accepted a position under Virchow as an assistant in the Pathological Institute in Berlin, where he remained for seven years. During this time he published many articles in Virchow's "Archiv für experimentelle Pathologie." He developed an interest at the Institute in the genesis of inflammatory conditions and published articles on tuberculosis of the choroid, inflammation and suppuration, and on mechanical hyperemia. Cohnheim was soon able to show that the pus of inflammatory conditions is derived from the blood.

In 1868, Cohnheim, who then was only twenty-eight years of age, was offered the newly established chair of pathology and pathologic anatomy at the University of Amsterdam. This he declined in favor of the chair of pathology at the University of Kiel. At Kiel, Cohnheim continued the work on the pathologic aspects of the

LECTURES

ON
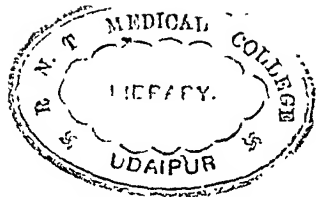
GENERAL PATHOLOGY.

A HANDBOOK FOR PRACTITIONERS AND STUDENTS.

BY

JULIUS COHNHEIM,

ORDINARY PROFESSOR OF GENERAL PATHOLOGY AND PATHOLOGICAL ANATOMY IN THE
UNIVERSITY OF LEIPSIG.

TRANSLATED FROM THE SECOND GERMAN EDITION

BY

ALEXANDER B. McKEE, M.B.Dub.,

CURATOR OF THE MUSEUM, ROYAL COLLEGE OF SURGEONS IN IRELAND.

WITH MEMOIR BY THE TRANSLATOR.

SECTION I.
THE PATHOLOGY OF THE CIRCULATION.

LONDON:
THE NEW SYDENHAM SOCIETY.

1889.

THE PATHOLOGY OF THE CIRCULATION*

By

JULIUS COHNHEIM

*Ordinary Professor of General Pathology and Pathological Anatomy in the
University of Leipsig*

SECTION I, CHAPTER IV

THROMBOSIS AND EMBOLISM

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But the locality to which the detached thrombi are transported is determined by anatomical conditions alone. Coagula from the venous side of the vascular system, *i.e.*, from the veins and the right heart, arrive in the *pulmonary arteries;* those from the arterial side, *i.e.*, from the left heart, the systemic arteries, and the pulmonary veins are conveyed into the *aortic system;* while those from the portal tributaries reach the *branches of the portal vein* in the interior of the liver. Not only are these general fundamental rules of transport throughout the vascular system prescribed by anatomical structure, but the special paths within any given portion of the circulatory mechanism are also so prescribed. The cause determining the entrance of the embolus into one artery rather than another can, at least in the case of larger plugs, be as a rule satisfactorily determined, and is to be sought in the direction of the embolized vessel with respect to the principal trunk, *i.e.*, in the angle at which it is given off, or in the relative calibre of the various lateral branches, or in some similar circumstance. Where the transport of the embolus appears to take place in opposition to anatomical laws, there are usually, as though to prove the rule, anomalies in the distribution of the vessels or in the heart. Thus I had quite lately an opportunity of observing a case of recent fatal embolism of one of the mid. cerebrals in a woman thirty-five years of age, where the valves of the heart, aorta ascendens, in short all the arteries from which an embolus might have been conveyed, were absolutely intact, while on the other hand an extensive thrombosis had occurred in the veins of the lower extremity. I had not, as you may suppose, at first the remotest idea of connecting the two conditions, till on more carefully inspecting the heart, I discovered a *foramen ovale* so

large that I could easily pass three fingers through it. I could not any longer reject the possibility that here a thrombus carried off from the *v. femoralis* had on its way through the heart passed from the right into the left auricle and thence into the mid. cerebral.* But the more one sees how perfectly the anatomical conditions are maintained in the course of such events, the more difficult, it seems to me, will it be to make up one's mind to indorse the opinion of certain authors that in cases, it is true of rare occurrence, emboli may be transported against the blood-stream, as for example, out of the *vena cava superior* into the hepatic vein.† It is an old experience and capable of verification at any moment that particles, say, of cinnabar, granules of mercury, or even plugs of wax may be very readily driven by the positive pressure of an injection-syringe from the *v. jugularis* into the hepatic veins; but this proves nothing with respect to the ordinary circulation, and I should, for my part, regard such an occurrence as very improbable, so long at least as the blood-stream through the portal vein is unimpeded.

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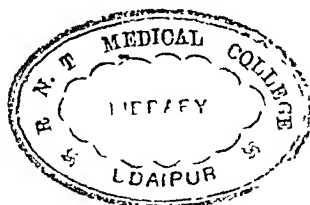
*Litten (Virch. A., IXXX, p. 381) describes a similar case, in which the *foramen ovale* being patent, a thrombosis of the right auricle was believed to have formed the starting-point for repeated embolism throughout the systemic vessels.

†Heller, D., Archiv f. klin. Med., vii, p. 127; Wagner, Allg. Path., p. 281.

1879

HENRI ROGER

DESCRIPTION OF THE MURMUR OF PATENCY OF
THE INTERVENTRICULAR SEPTUM

HENRI LOUIS ROGER

(Courtesy Faculty of Medicine, University of Paris.)
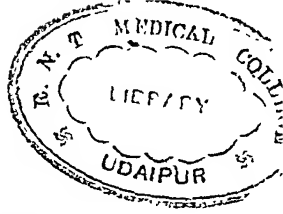
HENRI LOUIS ROGER

(1809-1891)

*H*ENRI ROGER was born at Paris on June 15, 1809. Following the example of two friends of the family, Drs. Guersant and Blache, he decided to become a physician. In 1833 he served an internship at the hospitals of Paris, and in 1847 he became physician to the hospitals of Paris and that same year he qualified for membership in the Faculté de Médecine. He served as physician to the Hospital for Sick Children from 1853 to 1874. He also was associated for twenty-two years as physician to the Sèvres Street Hospital and from 1862 until 1874 he was in charge of the Clinic in Paris.

Roger occupied a prominent position in the Association Générale des Médecins de France. He was president of the Société Centrale in 1872. In 1876 he was elected president of the Association Générale des Médecins de France and was constantly re-elected to this office until his death on November 15, 1891.

In 1839, the Société de Médecine et de Chirurgie de Bordeaux offered a prize for the best paper on the subject, ''To determine what progress has been made in diagnosis and treatment of diseases, particularly those of the lungs, heart, and great vessels, by means of auscultation, either mediate or immediate.'' Barth and Roger, who at that time were interns, submitted a paper for the award which had for its motto, ''If medicine is the most beautiful of the sciences, then auscultation is the most beautiful discovery of modern times.'' Although this paper did not receive the prize, which was awarded to Peyraud of Lyon, it did receive an honorable mention. It was the basis of a more extensive work by Barth and Roger on auscultation first published in 1841. This publication, greeted with enthusiasm by the medical profession, went through several editions and was translated into a number of foreign languages including the Scandinavian.

Roger's keen interest in auscultation combined with his skill as a pediatrician led him to discover an important anomaly of the septum, simple interventricular communication, later known as ''Roger's disease.'' In 1861, in performing a necropsy on the body of a young boy about twelve years of age, he found a malformation of the heart which consisted of failure of occlusion of the interventricular septum in its upper portion, without concomitant stenosis of the pulmonary artery. Necropsy also showed that the communication between the two ventricles would occur without cyanosis. After having listened to the heart sounds of thousands of children, Roger, with the aid of this pathologic discovery, was able to demonstrate that this lesion was characterized by the presence of a thrill and systolic murmur situated at the middle of the heart. The condition was not accompanied by any functional symptoms, cyanosis in particular and dyspnea being completely absent. Roger was also able to show that this congenital defect could exist without alteration of general health.

In 1879, Roger, after demonstrating this lesion in several instances, presented his observations to the Academy of Medicine. It is our privilege to present to our readers, in translation, Roger's observations entitled ''Clinical Researches on the Congenital Communication of the Two Sides of the Hearts, by Failure of Occlusion of the Interventricular Septum.''

CLINICAL RESEARCHES ON THE CONGENITAL COM-
MUNICATION OF THE TWO SIDES OF THE HEARTS,
BY FAILURE OF OCCLUSION OF THE
INTERVENTRICULAR SEPTUM*

By

HENRI ROGER

A MONG the congenital defects of the heart compatible with life and
perhaps a long one, one of the most frequent which I have encountered
(relatively frequent, absolutely rare) is the *communication between the
two ventricles because of failure of occlusion of the interventricular septum
in its upper portion.*

Of these cases (of which I have seen about a dozen), some *have cyanosis*
and some *do not;* and as there should be, in all cases, as a result of this
communication, a mixture of the two bloods, I have concluded with Louis
and Gintrac, who have best established this proposition, that the *morbus
ceruleus* does not arise from *the combination of arterial and venous blood,*
but that it is almost always attributable to a concomitant and likewise con-
genital lesion, *stenosis of the pulmonary artery,* the effect of this stenosis
being to impede and prevent the arrival of the blood in the lungs and conse-
quently the decrease of oxygenation.

Leaving aside the cases of cyanosis where the diagnosis should spring
up before the eyes, so to speak, but which are nevertheless complex and
where the anatomical conditions do not always reveal themselves to the
listener through abnormal sounds, I am going to speak here only of simple
cases, where there is a *communication of the two ventricles without morbus
ceruleus.*

This cardiac anomaly has no objective symptom which the eye can recog-
nize; it is dependent almost entirely on auscultation. After having listened
to thousands of children during forty years of special studies, and because
of incessantly repeated stethoscopic examinations, I am able, with the con-
trol of pathologic anatomy, to separate this anomaly of the heart from
other *malformations* and *diseases,* to establish its distinct clinical existence,
and to make of the *murmur* which characterizes it, a pathognomonic sign.

It was many years ago in village practice that I first recognized the
peculiar facts of auscultation; when I was called to consult about the

health of children, either for a recognized or suspected affection of the heart or for some other disease (not connected with the heart), I found, *among such children, almost all of them quite young,* a murmur of remarkable intensity, but with other characteristics which I thought unusual; what surprised me was that the murmur was almost the only sign of cardiopathy, and that it was accompanied by no other physical signs (save only the *purring thrill*), nor by any functional troubles indicating any lesion of the orifices or even of any signs of alteration of blood (since chloranemia is not at all a disease of early infancy, and the abnormal sounds heard in the very young are almost always on an organic basis).

What surprised me greatly while I had an opportunity to attend these children, to see them again, and to listen again at intervals more or less wide, was to find the same murmur after months or years with the same characteristics, without any appreciable alteration, without new physical signs, without alteration of general health, and *without cyanosis.*

I am going to cite three cases of these of which I have notes or precise memoranda. The first concerns a little boy of four months (Eugene L.) whom I saw several times and in whom I found at each examination the characteristic murmur unchanged, as was his general condition. I did not see him after the age of five years; his father, a physician, had set up practice in the province. In the second case I continued to see the little patient (Henry de B.) from the age of six months to his eighth year, when he was removed to Havre by his father, a customs collector. Several examinations, repeated at intervals of several years, showed identical results, an abnormal sound with a thrill which persisted without change, unaccompanied by any other sign of heart disease.

I have had the opportunity of observing a third patient during a much longer period of time, together with Blache: he was a Grammont-Caderousse, elder brother of the young duke who died of consumption and who was well known during his life for his eccentricities and after his death by the lawsuit of a physician, equally well known, to whom he had left his fortune. In this case, as in the others, I found a cardiac murmur having the aforementioned characteristics, and I found this murmur at each examination, persistent and unchanging, without visible damage to the organism. After about fifteen years I lost sight of this young man (the diseases of his youth required a specialist other than a pediatrician), until I read in the papers that a large steamer which was taking him to America had sunk in midocean. Later I learned from the family that the elder brother had not taken any better care of himself than had the younger, and that, although he was not robust (the mother had died of a tuberculous pleurisy), he had never appeared to suffer from organic heart disease.

These extraordinary cases, presented to my observation at long intervals, are difficult to interpret: I asked myself if this permanent *murmur* in the precordial region indicated an *endocarditis.* But if the abnormal

sound resulted from an alteration of the valves and orifices, why should it not show modifications of intensity and quality according to the constant progress of the affection, and further, why did not serious functional disorders finally manifest themselves?

On the other hand, the prolonged innocuousness of this supposed endocarditis was astonishing to me, and I asked myself rather if these little patients did not have a congenital anomaly of the circulation: if the murmur did not indicate a *communication between the two hearts* and if it was not produced by the passage of the column of blood across the orifice of the communication.[1] But if this supposition was correct, how did it happen that there was no *cyanosis* since it is generally admitted clinically that cyanosis is always the result of *communication between the two hearts and a result of the mixture of the two bloods?*

It was in the amphitheatre of L'Hôpital des Enfants, about 1861, that I saw the light and that I discovered the reason for these obscurities and apparent contradictions; in a young boy of 12 years, dead as the result of a comminuted fracture, I found at necropsy a *malformation of the heart,* which consisted of failure of occlusion of the interventricular septum in its upper portion, without concomitant stenosis of the pulmonary artery; in spite of the mixture of the two bloods which had resulted, neither the skin nor the tissues had been of blue color during life. It goes without saying that this malformation had been completely unrecognized by an entirely pardonable omission on a surgical service, the failure to listen to the heart.

Clinical circumstances had given me extraordinary facts, a chance occurrence of pathologic anatomy had offered the explanation. This necropsy, which showed that *communication between the two hearts could occur without cyanosis,* supplied the reasons for the facts that were little understandable until then; I did not doubt but what my former observations were of this same malformation which was shown to me so fortunately on the cadaver, and forthwith, applying this gift of morbid anatomy, I concluded that the *cardiac murmur, with particular characters* which I had discovered in my little patients, was a *pathognomonic sign* of the malformation.

It is necessary to explain in the same fashion cases similar to mine which clinicians have been able to find in children who were not cyanotic: they are mistaken as to the pathologic significance of the murmur heard,[2] attempting to connect them with an organic alteration of the orifices; it is not at all possible to throw these aside at the first observation, and even if they should be considered an anatomic anomaly, there is greater likelihood that it might be an acquired lesion, common and well known, rather than a congenital one, slightly known and wholly exceptional.

Thus are explained many other cases recalled further when I have been able to attend the patients during a long period of time and which have

[1] See Footnote 1, p. 633.
[2] See Footnote 2, p. 634.

furnished me proof that the failure of occlusion of the interventricular septum, when it is simple and not complicated by an anomaly, such as stenosis of the pulmonary artery, could exist and persist without compromising life or health.

Examples of the developmental anomaly, which I have been able to demonstrate as a clinical reality, are rare and are not found except by accident. It is probable, however, that (as is the case with newly described pathologic states) they will be found more numerous when, better known, they are studied with more care and observed more attentively. This was brought to my attention by the following:

On the thirtieth of last July a young man of seventeen came to ask for a certificate of physical fitness for employment as a postman; he was of small stature but of robust appearance and good health; he stated that he had never had a serious illness; he was not subject to bronchitis, palpitations nor breathlessness; he maintained that he could run as well as his friends. *He did not have cyanosis* and his color was ruddy and animated.

On applying the ear to the precordial region I heard forthwith a *harsh murmur* covering entirely the tic-tac of the heart, which could not be heard at any point. This murmur had its maximum intensity between the nipple and the sternum, somewhat stronger near the sternum; and from this center it was transmitted in all directions equally, diminishing by degrees according to the distance; and there was coincidentally a great *purring thrill*. Vertical dullness did not exceed the established limits by more than a centimeter (from the third rib to the fifth interspace). The cardiac impulse was strong and most clearly visible at the center noted, near the costochondral junction and not at the apex.

If we compare the symptoms of cardiopathy found in this young man with those which I have pointed out in the preceding observations and the general picture which I now portray, does not the diagnosis of *failure of occlusion of the interventricular* septum follow clearly from the comparison?

Nevertheless, this type of communication of the two hearts may not ordinarily be recognised at first; in the majority of cases the diagnosis is made by stages, and it may not be until after several observations that the observer, bearing in mind the true significance of the murmur, will be able to attribute it definitely to the malformation.

Let us review and further stress the *diagnostic data* furnished by *auscultation,* by the *comparison of local and general symptoms,* and also by *accessory considerations.*

These latter are by no means the least important for diagnosis; let us suppose, for example, that a physician has found a *cardiac murmur in an infant at the breast,* he will be in a great difficulty in determining its *clinical significance.*—Should he call it an *endocarditis,* and consequently

an organic murmur? But endocarditis, primary or secondary (a large source of abnormal sounds), is not a disease of the first years, and I do not recall having seen it before the second year. I have published a case of rheumatic endocarditis in a child of three years, and recently I have seen a report by Dr. Lecorche of endocarditis "a frigore" in a little boy of thirty-three months. As for torticollis and scarlatinal rheumatism, with which very young subjects may be afflicted, they do not have the tendency to involve the heart.—Should he call it an *inorganic murmur?* But *anemia,* which numerous causes (rachitis, tuberculosis, inanition) make quite common at the beginning of life, is never manifested by a cardiac murmur.

Thus, on the sole consideration of the *age* of the patient, the significance of the murmur is already presumptive, and one may announce *a priori* as a rule almost certain, that *a murmur in a suckling infant will be most often the result of an anomaly of the central circulation rather than of a disease.*

But it is *auscultation* which gives certainty to the facts which I have reported, and which I summarize from the text of my observations, the physical *signs* and above all *stethoscopic* signs, upon which I base the *diagnosis of patent ventricular septum.*

I have said that the sound, or rather murmur, indicating this communication of the two ventricles has *particular characteristics.*—It is generally remarkably *intense;* its *maximum* point is not at the apex (as in alterations of the auriculoventricular orifices), nor at the right base (as in stenosis of the aorta), nor at the left base (as in stenosis of the pulmonary artery) ; this maximum is in the superior third of the precordial region, and it is *median,* as is the interventricular septum itself.—It is *single* and greatly prolonged, beginning with systole and always covering the two normal sounds (a prolongation not usual with the murmurs of endocarditis) ; it replaces, or at least masks the natural tic-tac.—It is *stationary,* without transmission along the great vessels, whereas this transmission is often produced by pathologic murmurs arising from stenosis of the arterial orifices.—From the central point where it is at its greatest, it extends in every direction equally, and decreases in every direction, with the same regularity, according to the distance of the ear from its center.—The murmur is *coincident* with a strong impulse of the total mass of the heart, *without any very appreciable impact* at the apex, and with a *purring thrill,* of wide extent, which is in exact correlation with it.

With these *positive signs,* the murmur should be distinguished from that which results from a lesion of the cardiac orifices. The other signs, which are *negative,* should facilitate further the *differential diagnosis:* thus, in malformations of the heart, variations are not seen which diseases show, variations in cardiac dullness, more or less spread over the precordium (with or without bulging), according to variations in the degree of energy of the beat and impact with which the region is displaced, variations in the

intensity of tactile vibrations which are equally changeable; these differences are in accord with the various organic lesions of the heart and pericardium.

Indeed, a special symptomatology should correspond to these distinct anatomo-pathologic conditions: in *developmental failure* the lesion is single, identical with itself, changing slightly in the course of years; and the physical signs of it are few, permanent and, so to speak, unchangeable (with accompaniment of only slight difficulties of function which vary little). On the contrary, in *heart disease,* the lesions are multiple, inconstant, subject to change more or less rapid; these diseases (pericarditis, endocarditis and aneurysms) progress by periods; they have an acute stage, then a chronic one with or without exacerbations. Local and general symptoms of this complex and mobile picture show these successive phases and vary incessantly with the morbid process.

It is known that heart diseases, notably endocarditis, are very often *latent* in the first stage; the difficulties of circulation and respiration are often so slight at the beginning that they escape maternal vigilance; many children afflicted with chronic endocarditis with hypertrophy run, jump and play as if they were in perfect health, and without complaining afterward of palpitation or breathlessness, which, however, are actually present. It is the same in nearly all cases of patency of the interventricular septum. Thus, usually the physician is not consulted until more or less later, and on the occasion of some accidental illness. If he does not examine all of the systems, whether they are troubled or not (the most attentive pediatrician has the greater chance of being the best), if, impressed by the idea of the rarity of cardiac pathology in the early years of life, he neglects auscultation of the precordial area, he will necessarily pass by the true diagnosis and he will be ignorant of the murmur which would have revealed the cardiac lesion. If, on the contrary, he remembers to use his ear, he will immediately perceive the abnormal sound which will show at least that the diseased organ is the heart   Possibly he will mistake the significance of this murmur and will believe that an endocarditis is present (this is the only error that should be possible); but such an error, until then inevitable, he will correct in good time by carefully comparing the characteristics which differentiate the murmur of patent interventricular septum and murmurs belonging to the orifices, characteristics which I have recognized and of which I have shown the clinical value.

The precision of diagnosis, of interventricular patency which I believe I have made hereafter easy, is concerned especially with *prognosis,* and it is interesting to know that an infant, especially if he is very young, has a *congenital anomaly* of the heart, through which life will not be directly endangered, and not an *organic disease.* Without any doubt, communication of the two ventricles is a serious anomaly of circulation and of aeration; congenital and consequently irreparable, it has its own seriousness.

Nevertheless, this seriousness is less than that of phlegmasias of the pericardium and endocardium, where the damage is double, by accidents perhaps quickly fatal in the acute stage, and by those of the chronic period where progress is disastrous and the outcome always fatal. Endocarditis, less serious than pericarditis, is curable in theory and it sometimes heals, but in fact when the resolution of the inflammatory products delays more than one or two years, when secondary lesions of the orifices are advanced, healing is not to be hoped for; one may beguile himself in vain that with the changes brought about in the organism by the climacteric period these organic changes will correct themselves; time, far from diminishing the disease, makes it worse; and it is known that the nature of these organic affections is unceasing progression with lesions which were at first local becoming generalized. It is a pathologic circle that widens more and more.

In these diseases the peril is at least as great as in simple malformations and much more acute: I have seen a few infants recover from an acute endocarditis, but I have never seen any stricken with chronic endocarditis who reached mature years. The chances of survival are actually greater in patients with an anomaly than in those with heart disease: with the latter, young patients cannot hope to live more than an average of ten years; with the former, the average is two or three times as long.

Several subjects whom I have been able to observe, I have attended for periods of *five, twelve and fifteen years;* these children have grown like others, not one has died prematurely; and except for a tendency to pulmonary catarrh the general health has not been compromised in a single one by reason of the cardiac malformation. It is certain that they should have lived well beyond the time when I last saw them, because at the time of the last visit they appeared stronger and in better health than at the first.—The young girl whose history was reported in our *Traité d'auscultation* was sixteen years old and was in good health.—The boy whom I saw recently was *seventeen* and he appeared very capable of carrying out the laborious work of postman.

Finally, now for some twenty years, I have occasionally visited as physician a woman whose children I attended from early ages and who contracted scarlet fever from one of her children: she had always been in excellent health and had never complained of cardiac difficulties. On auscultation I was greatly surprised to hear a murmur, with characteristics which struck me as very peculiar, which I thought immediately was the result of a congenital anomaly. I asked her if physicians had ever found *anything wrong with her heart,* and she told me that Guersant the Elder (the famous pediatrician, who was my first master in infantile pathology) had recognized in her a few days after birth, a cardiac malformation.— This woman has now passed her fiftieth year; her health continues to be perfect and she is the mother of four children. I went to see her again after several months to pay my respects on the marriage of her elder

daughter, and I asked permission to listen again (by virtue of her being such an unusual woman) and I found again in the precordial region the same murmur as previously, with all of the characteristics of the *murmur of patent interventricular septum.*

It can be understood that there is no direct treatment for the cardiac malformation: the pathologic state of the heart existing before birth and consisting of an arrest of development is not susceptible to favorable changes, either by spontaneous evolution or by medical or surgical intervention; there is then, in these unavoidable conditions, nothing to attain, for the present or in the future, by medicine or reparative surgery. I say only, to lighten the severity of this conclusion, that the congenital lesion itself does not, like a disease, progress; if one may not hope for the diminution of a disease, one does not at least have to fear an augmentation, and danger will not come unless morbid changes supervene and complications occur.

The prevention of complications is by means of *hygiene:* it is this which will furnish the means of attenuating the effects of the anomaly in the circulation, and of prolonging life, not only to the average limit but even beyond. Children who have been found to have patent interventricular septum should be, as the saying goes *mollycoddled;* it is necessary to preserve them with vigilant care from *cold*, which engenders catarrh and rheumatic affections, and to prevent diseases of the respiratory tract which are, with them, more serious, disorders of the respiratory function aggravating the circulatory disorder and vice-versa. In the second period of childhood as well as in the first, it is equally necessary to observe precautions to prevent as far as possible any broncho-pulmonary accident.

It is necessary to follow this preventive system for a long time and perhaps never to depart from it, because these patients, whether adolescents or adults, cannot live as does everyone else and do with impunity what normal and healthy people can do without damage.

Everything which strains the heart, accelerates the beat, augments the activity and tends, after a time, to produce secondary hypertrophy, should be avoided. These recommendations are not addressed, it should be well understood, to the very young. While they are in the cradle or the maternal knees and arms, they are preserved by their sedentary life from circulatory difficulties and for this reason from hypertrophy, and with them, in the earliest years, the cardiac anomaly is wholly *latent* (except to the physician). For older children it is necessary to interdict excessive play, violent exercises and above all, gymnastics (beloved by mothers who wish to make little athletes of their children). The same interdictions hold for adolescents, and for those who have arrived at adult age one should give the advice (easy to give, difficult to follow and almost always unheeded) to abstain from all excesses; one should endeavor to make them understand that their health is their reward, and that prolongation of life

will recompense their moderation: this is the hygienic precept of the poet—
"He who would ride far should spare his horse."

In a word, although prudence is necessary in each case, there is no advantage in treating this malformation of the heart, with regard to hygiene, as if it were a disease, properly speaking.

Formerly, consultations for organic affections of the heart ended with this last prescription: "Avoid emotions of mental distress."—This traditional formula, even more used today, has always seemed to me naive as well as banal: strokes of bad fortune, painful emotions, deep chagrin, come to everyone without being sought; the more excitable the nervous system, the more it is disturbed. Whether he is sick or well, man is not the master of his emotions because he does not command the pleasant or unpleasant events of which he receives the impression: he is not able to *avoid* one or another at his will, and least of all, by means of a medical prescription.

The *service* which an accurate diagnosis of patent interventricular septum renders the patient affected is none the less real because it is *indirect.* As much as it is necessary that the practitioner should guide heart disease in infants and adults with energetic and persevering treatments, it is equally useless and even harmful to give such medication for cardiac malformations; as much as action is necessary in the first case, by so much is inaction better in the second.

Actually, all of the resources of therapeutics (and they are unfortunately quite few and altogether impotent) should be used to combat cardiac phlegmasias at their beginnings (local bleeding, resolving and soothing applications, revulsives, vesicants, etc.); to moderate them and to obtain a cure, which is, however, unusual. And later, in spite of the development of organic lesions, in spite of the difficulty, not to say the impossibility, of resolving or impeding secondary degeneration, it is necessary, by rational medication incessantly continued and varied, and otherwise, to meet therapeutic indications shown by the local lesion, by the general difficulties which arise from it and by the complications which take place on every side. But what is the opportunity, what is the utility of a similar treatment given for a malformation which is known to be irreparable?

With *digitalis,* for example, what good can be expected from this medicament so valuable in the course of cardiac affections and so necessary in exacerbations, of this indispensable medicament which practitioners (even those who deny its good effects on theoretical grounds) will not be without? Limited in its salutary action, it is only positively useful in certain phases of disease; it is incapable of changing a permanent anatomopathologic state like malformation; with this state that continues indefinitely, one should have a remedy the effectiveness of which should be equally inexhaustible: but it is known that digitalis, effective as it is when given temporarily, becomes harmful when its use is prolonged, the therapeutic effects of the herb being replaced by the toxic ones.

What should we say of the application of cautery to the precordial region? Formerly it was the classical treatment for cardiac affections; many years ago, during my internship in the Hôtel-Dieu, I recall that the famous Récamier (an intrepid and inventive therapeutist, who was never disarmed or discouraged by the most desperate case) had the custom of prescribing in such a case, four cauterizations of the precordial region, in order, he said, to circumvent the disease on all sides. Did the disease thus attacked yield? The majority of physicians of that time cherished the illusion, and their convictions were little disturbed by contradictions of the autopsy.

When patent interventricular septum is recognized such *energetic* medication is not appropriate; it is absolutely proscribed by virtue of the diagnosis. It is irrational to use cautery, vesicants or any other cardiac remedy to treat an incurable congenital defect. With these irrevocable conditions, what should the physician do? He should attempt to retard, by means of the resources of hygiene, the unfortunate effects of nature's error and to attenuate the subsequent influence of the local lesion on the general health; his power does not extend very far and the hope of cure is impossible; he should not torment and fatigue the organism by disquieting medication, inefficacious as well as irrational and finally harmful. It is said with reason that the progress of diagnosis contributes to that of treatment; here, against an insurmountable obstacle, progress is at a standstill; the obligation of the therapeutist is to forbear and to efface himself before the hygienist. An exaggerated medication surely has more dangers than pure expectation: *primo non nocere*, is the adage of ancient medicine and it will be the last conclusion of this work.

Footnotes

(1) By stating several facts known to science, I have completed, *a posteriori*, my response to appropriate observations presented to me after reading this paper.

In patency of the interventricular septum, I do not see *any other cause for the murmur* than the *flow of blood* from left to right *across the communication*. The mixture of arterial and venous blood which must take place is scarcely contestable, when we recall the differences of pressure which exist, according to the experiments of Marey, between the two ventricles, the force of contraction of the left is equal to 128 millimeters of mercury, that of the right, only 25. In the cases in which stenosis of the pulmonary artery is present, the right ventricle becomes hypertrophied and the left atrophied, and it is possible that with the relationships being reversed, blood should cross the abnormal opening in the septum with the murmur from right to left. But with these complex conditions it is difficult to know what the acoustic phenomena should be and what their mechanism is without considering that stenosis of the pulmonary artery gives rise to a murmur which adds peculiarly to the difficulties of a precise and complete diagnosis.

Am I deceived of the significance of the *murmur* and of its clinical value? Could it be the sign of *persistence of the foramen ovale* or of the *ductus arteriosus* and not of interventricular patency? This opinion does not appear at all tenable to me, the contraction of the auricles being impotent to force the blood from one auricular cavity to the other with enough force to produce a murmur. Moreover, the isolated existence of

one or the other of these congenital abnormalities is quite rare, and they are generally surpassed by much more serious congenital lesions with more accentuated signs; thus stenosis of the pulmonary artery, with the cyanosis which it produces, is, so to speak, the forced accompaniment of patent foramen ovale, since this patency is almost always the effect of the arterial obstruction. In about thirty of the cases of patent foramen ovale cited by Peacock in his scholarly work *(On Malformations of the Human Heart)* there were not more than four or five in which this malformation *appeared to* be simple; I say *appeared*, because in the majority there was cyanosis, which means the likelihood of some other concomitant cardiac lesion.

Of all of these cases, only one (observed by the physician of St. Thomas's Hospital) is of some value from the point of view of *auscultation:* in a young girl who died of consumption after having showed also symptoms of heart disease, there were found at necropsy, in addition to tuberculous changes, a mitral stenosis with slight hypertrophy of the right ventricle, and a *persistence of the foramen ovale,* the opening about the size of a shilling. During life she had had neither *maladie bleue* nor any murmur anywhere in the precordial region.

Dr. Ernest Labbee presented to the Anatomical Society in 1865 a heart in which *persistence of the foramen ovale* had been shown; the patient afflicted with this slight malformation was an old man who had never had any cardiac difficulties and in whom auscultation, frequently done, did not reveal any *murmur* or any other alteration of the normal heart sounds.

Dr. Duroziez, an exact and conscientious observer who has consecrated long years to the special study of vascular sounds and cardiac murmurs, was asked if persistence of the foramen ovale could give rise to abnormal sounds: he said that he had never found an example and that the rare observations which had been cited did not appear demonstrative to him.

As for me, I have found only *one example,* in the amphitheatre of l'Hôpital des Enfants, of *persistence of the foramen ovale* in the absence of any other cardiac lesion; during life this anomaly had not given rise to any evidence of circulatory functional difficulty nor to any auscultatory sign.

I oppose the same considerations and the same facts to the theory which seeks to assign the above described *murmur* to an origin in the *ductus arteriosus* and to the passage of blood through this patent duct for a physical cause. Here, further, positive observations are absolutely wanting, and no mention is made of a murmur indicating this malformation in any of the numerous cases reported by the writers *(vid.* Peacock, *loc. cit.,* and the admirable *Traité clinique* of Bouillaud).

There are, on the contrary, *two negative observations* reported to the Biological Society by Luys and by Duroziez. The first concerns a woman, fifty-eight years of age, in whom was found at autopsy a direct communication between the aorta and the pulmonary artery by means of a *persistent ductus arteriosus* which would admit the tip of the ring finger; hypertrophy of the right ventricle was also present. *No abnormal sound had been heard in the region of the heart.* The second case, a man forty years of age, was found to have an hypertrophy of the right ventricle with dilatation of the pulmonary artery and relative atrophy of the left ventricle: *the ductus arteriosus was persistent.* Auscultation, practiced frequently, *did not reveal any cardiac murmur.*

Without denying the possibility of the existence of a cardiac murmur in a case of patency of the foramen ovale or the ductus arteriosus, I consequently affirm the diagnostic value of the *murmur* which is *almost always* the sign of *patency of the interventricular septum.*

(2) In the eighth edition (1874) of *Traité d'auscultation,* there is recorded in a note (p. 476) an observation collected by my collaborator and perpetual friend, M. Barth, where he caught a glimpse of what I see clearly today: ''In a young girl of sixteen

years, there was found over the heart an intense sonorous *murmur* with markedly pronounced *purring thrill;* the murmur had its maximum at the level of the *bifurcation of the pulmonary artery* and was diminished according to the distance from this point. What might be the cause? Could it be the *persistence of the ductus arteriosus* or rather a communication of the two hearts because of patency of the interventricular septum? The lesion appeared to us to be congenital. The girl was in good health otherwise and could sing without difficulty: *we did not note cyanosis.*"

Conclusions

1. A *developmental defect of the heart occurs* from which *cyanosis* does not ensue in spite of the fact that a communication exists between the cavities of the two ventricles and in spite of the fact that admixture of venous blood and arterial blood occurs. This congenital defect, which is even compatible with a long life, is a simple one, without the association of congenital pulmonary stenosis. It comprises a defect in the interventricular septum.

2. It is necessary to differentiate this anomaly of the heart, which I have recently been the first to study clinically, not only from other malformations, but particularly from acquired disease of the heart. Its presence is revealed only by auscultation, through a physical sign with definite characteristics: this consists of a long loud *murmur* (resulting from the passage of blood through the opening in the interventricular septum and directly into the pulmonary artery or the aorta, the location of which is frequently abnormal in these cases). This murmur is unaccompanied by other murmurs, begins with systole and is so prolonged that it entirely occupies the period of the natural tic-tac of the normal heart sounds. Its point of maximal intensity is not at the apex (as in the case of lesions of the auriculoventricular orifices), nor at the base on the right side (as in stenosis of the aortic orifice), but over the upper third of the precordial area. It is mainly medial in location like the septum itself, and from this focal point diminishes uniformly in intensity as the stethoscope is moved over the chest. The murmur is not propagated into the vessels. It coincides with no other sign of organic disease with the exception of the *harsh thrill* which accompanies it. This murmur is *the pathognomonic sign of a defect in the interventricular septum.*

3. The differential diagnosis of this malformation (up to the present time either unrecognized or confused with other congenital or acquired lesions) will from now on be rendered simple by careful comparison of the physical signs. These signs vary in number, location, and character in heart disease when structural alterations are multiple, progressive, and changing, while the murmur under discussion, like the permanently fixed lesion responsible for its occurrence, remains unaltered for an indefinite period of time. The same statement holds true when comparing this murmur with the *signs of functional disturbances;* such signs vary with the changing episodes of heart weakness, and are entirely different in their acute or chronic charac-

teristics from the unaltered signs of a defective interventricular septum which change inappreciably over the years and increase only very gradually and almost without detection.

4. The consideration of the age of the patient is a noteworthy point in the diagnosis; endocarditis, for example, is rarely seen in infancy, before the age of two years, and furthermore, the anemia of very young children is very seldom associated with a heart murmur. Thus, a *murmur in a nursing infant* is almost always a definite indication of an *anomaly* of the heart or great vessels.

5. The *prognosis* is generally less significant in the abnormality described than in other structural diseases of the heart, in which the danger for children is greater and occurs sooner, permitting hope for not much more than another decade of life. In spite of the existence of an uncomplicated defect of the interventricular septum, patients may attain or even surpass the average span of human life.

6. A definite diagnosis usually demands an active sustained program of *treatment* in heart disease. But, if a congenital anomaly of the heart exists, vigorous treatment is of no avail and may even be harmful. To show, thanks to the accuracy of diagnosis, when to act in one and when to withhold treatment in another, is to be of service not only to physicians but also to patients.

COMMUNICATION CONCERNING CONGENITAL PATENCY OF THE INTERVENTRICULAR SEPTUM*

By

HENRI ROGER

In the paper which I read to the Academy last October 21, I sought to establish the distinct clinical existence of a cardiac anomaly not recognized up to the present, and confused, sometimes, with other congenital anomalies, sometimes with heart disease properly speaking, namely, *congenital communication of the two hearts by patency of the interventricular septum:* I presented a differential diagnosis which arises almost exclusively from auscultation and I stated that a *special cardiac murmur* was the *pathognomonic sign of this malformation.*

Two objections have been made: the first upon the *anatomical site of the murmur* which I attributed to the passage of blood across the perforation of the septum in its superior portion, that it might also as well (they tell me) be produced by the passage of blood across the foramen ovale or ductus arteriosus. For the other, it has been objected that my clinical description was not based upon a sufficient number of necropsies.

For completing my answer to these objections, I request permission of the Academy to read, in summary, an *autopsy report* which Dr. Gaston Decaisne published in *Progrès Médical,* under the title, "Congenital communication of the ventricles of the heart," presented to the Anatomic Society in July 1877.

"In a little girl of 26 months (D—) who entered the Hôpital des Enfants on the service of M. Bouchut, who did not appear to have any heart disease and who was *not at all cyanotic,* auscultation revealed *an intense systolic murmur* occupying the entire precordial region, it was heard on the right side of the chest and even in the posterior thoracic region. The maximum of this murmur was over the *base and middle portion of the heart.* There was considerable bulging with a *purring thrill.* The pulse was 102 per minute, moreover it was regular, not intermittent. Cardiac dulness was not appreciably enlarged.

"In the absence of general symptoms the diagnosis of acute cardiac affection could be discarded and the existence of a *congenital lesion* was admitted. But where was the lesion? The *absence of cyanosis* excluded

stenosis of the pulmonary artery. There was nothing to choose from except *coarctation of the aorta* and *patent foramen ovale.* Finally the last diagnosis was accepted, with reservations however.

"The child died about six weeks later, after some pulmonary mishap.

"*Autopsy.*—The *heart* is not hypertrophied. The *aorta* and *pulmonary artery,* by no means narrowed, are perhaps slightly *dilated,* but their valves are normal. The mitral and tricuspid valves are likewise. The ventricular walls show no alteration but *in the upper portion of the interventricular septum,* beneath the mitral valve, is found an *orifice* which establishes a *communication between the two ventricles.* On the side of the left ventricle the opening is sinuous; on the side of the right ventricle it is on the contrary curved and prominent. The endocardium at this level is whitish, thickened and opaque. The *foramen ovale is obliterated.*"

In this important observation which is interesting and rare and which does not lack the control of pathologic anatomy, was found the congenital lesion of the interventricular septum which I have separated from other malformations, the particular murmur which characterises it, and the coincidence of positive and negative signs of which I have shown the clinical picture. This observation gives me proof on all points; I wish to thank my young and distinguished colleague who was so kind as to send it to me.
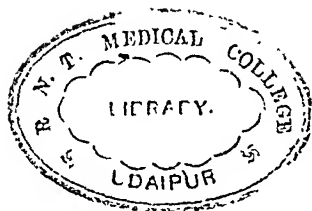
1879

WILLIAM MURRELL

INTRODUCTION OF NITROGLYCERIN IN THE
TREATMENT OF ANGINA PECTORIS

WILLIAM MURRELL

(1853-1912)

ILLIAM MURRELL was born in London on November 26, 1853, the son of William Kenrick Murrell, a lawyer. He received his primary education at Murray's School in Wimbledon and his academic training at University College, London, where he was awarded the William Sharpey Physiological Scholarship and where he became a demonstrator in physiology. In 1874 he qualified for the licentiate of the Society of Apothecaries, and in 1875 he qualified for membership in the Royal College of Surgeons, England, and obtained the licentiate of the Royal College of Physicians of London. In 1877 he passed the qualifications for membership in the Royal College of Physicians. That year, also, he was appointed medical registrar to the Westminster Hospital, having served previously as a teacher of histology. He studied medicine at the University of Brussels and in 1879 received from that institution the degree of Doctor of Medicine. He was elected a fellow of the Royal College of Physicians in 1883.

Murrell devoted much of his lifetime to the study of pharmacology and therapeutics. Early in his career he came under the influence of the British physiologist, Sydney Ringer (1835-1910), who at that time was making extensive experimental researches on the action of various drugs on living protoplasm. At this period the leading physicians who served on the staffs of hospitals did not bother with the details of the prescribing of drugs, but left the task to house physicians. They also showed no concern about either the physiologic or the pharmaceutic action of drugs. Hence, they did not bother to encourage elementary instruction in these subjects or to avail themselves of chances to accomplish the fundamental research which was badly needed.

Murrell often expressed disappointment about such a state of affairs, and even though his colleagues discouraged him, he continued his pharmacologic studies.

In 1879 appeared his first independent publication, "Nitroglycerine as a Remedy for Angina Pectoris." This important and original observation we are including in our classics. When this paper was published, Murrell was lecturer on practical physiology at the Westminster Hospital and was assistant physician to the Royal Hospital for Diseases of the Chest. In 1883, he was appointed assistant physician, and fifteen years later became physician, to Westminster Hospital.

Murrell's best known work is his "Manual of Pharmacology and Therapeutics," first published in 1896. His interest in toxicology was paramount at a time when the medical profession showed little interest in the subject. Nevertheless, his book, "What to do in the Case of Poisoning," went through eleven editions before his death.

Murrell was also instrumental in exposing the scandalous massage establishments which at one time flourished in the West End of London. He tried, without too much success, to place massage on a scientific basis and in 1886 published his work, "Massotherapeutics, or Massage as a Mode of Treatment." However, it was many years before the profession realized the importance of his early efforts in this field.

In later years, Murrell took an active interest in bacteriotherapeutics.

At the time of his death, he was a member of the Faculty of Medicine of the University of London. In 1881 he was elected a laureate of the Academy of Medicine of Paris. In 1887 he served as one of the vice-presidents of the International Medical Congress, which was held that year in Washington, D. C. For some years, he served on the medical staff at the Paddington Green Children's Hospital, where he later became senior physician.

For several months previous to his death, Murrell had suffered from heart disease. In the spring of 1912, dropsy supervened and he was obliged to go away for a rest. As soon as he felt better he returned to his medical practice. Soon he had a second breakdown. He suffered from extreme hypertrophy and dilatation of the heart with complications, which resulted in his death on June 28, 1912, at the age of fifty-eight.
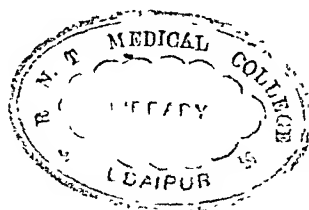
NITRO-GLYCERINE AS A REMEDY FOR
ANGINA PECTORIS*

By

WILLIAM MURRELL, M.R.C.P.

Lecturer on Practical Physiology at Westminster Hospital, and Assistant-Physician to the Royal Hospital for Diseases of the Chest

SOME TWENTY years ago a controversy took place in the pages of the *Medical Times and Gazette,* on the properties, physiological and therapeutical, of the substance known to chemists as nitro-glycerine. The discussion was opened by Mr. A. G. Field, then of Brighton, who described in detail the symptoms he had experienced from taking two drops of one per cent solution of nitro-glycerine in alcohol. About three minutes after the dose had been placed on his tongue, he noticed a sensation of fulness in both sides of the neck, succeeded by nausea. For a moment or two there was a little mental confusion, accompanied by a loud rushing noise in the ears, like steam passing out of a tea-kettle. He experienced a feeling of constriction around the lower part of the neck, his forehead was wet with perspiration, and he yawned frequently. These sensations were succeeded by a slight headache and a dull heavy pain in the stomach, with a decided feeling of sickness, though without any apprehension that it would amount to vomiting. He felt languid and disinclined for exertion, either mental or physical. This condition lasted for half an hour, with the exception of the headache, which continued till the next morning. These symptoms Mr. Field described as resulting from a single dose of one-fiftieth of a grain. Thinking that possibly he might be unusually susceptible to the action of the drug, he induced a friend to take a dose. The gentleman experienced such decided effects from merely touching his tongue with the cork of the bottle containing the nitro-glycerine solution that he refused to have anything more to do with it. A lady suffering from toothache, on whose tongue Mr. Field placed about half a drop of the same solution, experienced a pulsation in the neck, fulness in the head, throbbing in the temples, and slight nausea. The toothache subsided and she became partly insensible, disliking much to be aroused. When fully sensible she had a headache, but the tooth-ache was gone. Another of Mr. Field's patients, a stout, healthy young woman, accidentally swallowed a small piece of lint dipped in the nitro-glycerine, whilst being applied to a decayed tooth. In about five minutes,

after feeling giddy and sick, with headache, she became insensible. Her countenance, naturally florid, was unaltered, breathing tranquil, pulse full, and rather quickened. She recovered in about three minutes, after the administration of a stimulant. Some headache was complained of, but the toothache was gone. Mr. Field, in conclusion, offered some suggestions as to the therapeutical uses of the drug, and stated that he had not met with a single well-defined case of neuralgia or spasmodic disease in which it had failed to afford some relief.

This paper was followed by a letter from Dr. Thorowgood, in the main confirmatory of Mr. Field's observations. He, after taking a small dose, experienced "a tensive headache over the eyes and nose, extending also behind the ears, and soon followed by a tight, choking feeling about the throat, like strangulation. Neither loss of consciousness nor nausea was experienced, and a walk by the sea soon did away with the unpleasant feeling."

These statements did not long remain unchallenged, their accuracy being called in question by Dr. George Harley, of University College, and Dr. Fuller, of St. George's. Dr. Harley, having obtained some nitro-glycerine of the same strength as Mr. Field's, commenced his observations by touching his tongue with the cork of the bottle containing the solution. He experienced "a kind of sweet and burning sensation, and soon after a sense of fulness in the head, and slight tightness about the throat, without, however, any nausea or faintness." After waiting a minute or two these effects went off, and Dr. Harley was inclined to think "they were partially due to imagination." Determined, however, as he says, to give the drug a fair chance, he swallowed five drops more, and as this did not cause any increased uneasiness, he took, in the course of a few minutes, another ten drops of the solution. Being at the time alone he became alarmed lest he should have taken an over-dose, and very soon his pulse rose to above 100 in a minute. The fulness in the head and constriction in the throat were, he thought, more marked than after the smaller dose. In a minute or two the pulse fell to 90, but the fulness in the head lasted some time, and was followed by a slight headache. To two medical friends Dr. Harley administered respectively twenty-eight and thirty-eight drops in divided doses without the production of any symptoms. Some pure nitro-glycerine was then obtained, and of this Dr. Harley took, in the course of a few minutes, a drop, equivalent to a hundred drops of the solution previously employed. The only symptoms produced were a quickened pulse, fulness in the head, and some tightness in the throat; but as these passed off in a few minutes, Dr. Harley considered that they were probably the effects of "fear and imagination." On a subsequent occasion he took, in the course of three-quarters of an hour, a quantity of the nitro-glycerine solution equivalent to 199½ drops of the solution used by Mr. Field, with the production of no more dis-

agreeable symptoms than those he had experienced in his former trials. The quickening of the heart's action he ascribed to fear, but the head and neck sensations were, he considered, "too constant to be attributed to the same cause," although he thought they were exaggerated by the imagination. Dr. Harley, in conclusion, states that he experimented on ten different gentlemen with nitro-glycerine solution, obtained from four different sources, without witnessing any dangerous effects when administered in the above doses; but he adds that, if taken pure, great caution should be used.

. . . .

In a second communication to the same journal Mr. Field reasserted the correctness of his observations, and maintained that a reasonable explanation of the very different results obtained by different observers might be found in the great variation in strength to which the drug is liable. He considered, too, that the conditions under which the drug was taken had much to do with its action. When the system is worn out by fatigue, he says, it is more likely to act powerfully than when taken under less unfavourable conditions. On the occasion of taking the dose which produced in him such startling effects, his nervous energy had been impaired by an unusually hard day's work. He found that under more favourable conditions he could take the same dose with production of nothing worse than headache. Having in his experiments on himself experienced the greatest variation in the strength of different specimens of nitro-glycerine, he was disposed to think, on reading the account given by Dr. Fuller and Dr. Harley, that they had used a less powerful agent. He accordingly called on Dr. Fuller, and induced him to take a dose of the solution he had used, but, to his surprise, he experienced little beyond headache. On the same day Mr. Field administered to a hospital patient suffering from hemicrania two drops of the solution. In about a minute he became pallid, felt sick and giddy, his forehead was covered with perspiration, and he sank on the bed by which he was standing almost unconscious, his pulse failing so as scarcely to be felt. After the administration of a little ammonia the circulation became more vigorous, and in twenty minutes there was a marked diminution of the pain, and he experienced a great desire to sleep, a luxury of which his sufferings had almost deprived him on previous nights. Mr. Field administered small doses of the drug to several other people, all of whom were distinctly affected by it.

. . . .

Being greatly interested in this curious controversy, and being quite at a loss to reconcile the conflicting statements of the different observers, or arrive at any conclusion respecting the properties of the drug, I determined to try its action on myself. Accordingly I obtained some one per cent solution. One afternoon, whilst seeing out-patients, I remem-

bered that I had the bottle in my pocket. Wishing to taste it, I applied the moistened cork to my tongue, and a moment after, a patient coming in, I had forgotten all about it. Not for long, however, for I had not asked my patient half a dozen questions before I experienced a violent pulsation in my head, and Mr. Field's observations rose considerably in my estimation. The pulsation rapidly increased, and soon became so severe that each beat of the heart seemed to shake my whole body. I regretted that I had not taken a more opportune moment of trying my experiments, and was afraid the patient would notice my distress, and think that I was either ill or intoxicated. I was quite unable to continue any questions, and it was as much as I could do to tell him to go behind the screen and undress so that his chest might be examined. Being temporarily free from observation, I took my pulse and found that it was much fuller than natural and considerably over 100. The pulsation was tremendous and I could feel the beating to the very tips of my fingers. The pen I was holding was violently jerked with every beat of the heart. There was a most distressing sensation of fulness all over the body and I felt as if I had been running violently. I remained quite quiet for four or five minutes and the most distressing symptoms gradually subsided. I then rose to examine the patient, but the exertion of walking across the room intensified the pulsation. I hardly felt steady enough to perform percussion and determined to confine my attention to auscultation. The act of bending down to listen caused such an intense beating in my head that it was almost unbearable and each beat of the heart seemed to me to shake not only my head, but the patient's body too. On resuming my seat I felt better and was soon able to go on with my work, though a splitting headache remained for the whole afternoon. Were my symptoms due to nervousness or anxiety? Certainly not. I will not say that I discredited Mr. Field's observations, but after Dr. Harley's positive assertions I certainly did not expect to obtain any very definite results from so small a dose. Moreover, at the moment of the onset of the symptoms I was engaged in the consideration of another subject and had forgotten all about the nitro-glycerine. I did nothing to intensify the symptoms, but, on the contrary, should have been only too glad to have got rid of them. The headache, I can most positively affirm, was anything but fancy. Since then I have taken the drug some thirty or forty times, but I never care to do so unless I am quite sure that I can sit down and remain quiet for a time, if necessary. It uniformly produces in me the same symptoms, but they are comparatively slight if I refrain from moving about or exertion of any kind. The acceleration of the pulse is very constant, although sometimes it amounts to not more than ten beats in the minute. The temperature remains unaffected. The pulsation is often so severe as to be acutely painful. It jerks the whole body so that a book held in the hand is seen to move quite distinctly at each beat of the heart.

The amount of pulsation may be roughly measured by holding a looking-glass in the hand and throwing the reflection into a dark corner of the room. Before taking the drug the bright spot may be kept steady, but as soon as the pulsation begins it is jerked violently from side to side. I have taken all doses from one minim to ten, sometimes simply dropped on the tongue, at others swallowed on sugar or in water. I have not ventured to take more than fifteen minims in a quarter of an hour. Once or twice a ten minim dose has produced less pulsation than I have experienced at other times in a single drop; but then with the larger quantity one is careful to avoid even the slightest movement. After a five minim dose I usually experience a certain amount of drowsiness—a lazy contented feeling, with a strong disinclination to do anything.

Thinking there might be individual differences of susceptibility to the action of nitro-glycerine, I have laid my friends and others under contribution and have induced as many as possible to give it a trial. I have notes of thirty-five people to whom I have administered it, twelve males and twenty-three females; their ages varying from twelve to fifty-eight. I find they suffered from much the same symptoms as I did, although it affects some people much more than others. Of the numbers above quoted, only nine took minim doses without experiencing decided symptoms. Women and those below par are much more susceptible to its action than are the strong and robust. A delicate young lady, to whom, adopting Mr. Field's suggestion, I administered it in drop doses for the relief of neuralgia, experienced very decided effects from it, each dose producing a violent headache lasting from half an hour to three hours. A married woman, aged thirty-five, took one minim with very little inconvenience, but was powerfully affected by two. She was obliged to sit down after each dose and was positively afraid to move. It made her hot and caused such a beating in her head that she had to support it with her hands. She experienced a heavy weight on the top of the head and also a sharp darting pain across the forehead, which for a moment or two was very painful to bear. A friend, who for some days took four drops every three or four hours, informs me that at times it affected his head "most strangely." The pulsation was very distressing and often lasted an hour or more, being intensified by moving. It has relieved him of an old-standing facial neuralgia, and he is enthusiastic in its praise. A young woman, aged twenty-nine, complained that after every dose of the medicine—one minim—"it seemed as if the top of her head were being lifted off," and this continued sometimes for five minutes and sometimes longer. The medicine made her bewildered, and she felt sick. A patient with a faint apex systolic murmur was ordered one minim in half an ounce of water four times a day. He took two doses, but it caused "such a beating, thumping, hot pain" in his head that he was unable to continue it. A young man who was given nitro-glycerine in mistake for

phosphorus said it made his temples throb, and he could see his pulse beat so distinctly that he was frightened. It caused a burning and flushing in his face, and "took every bit of strength away." This would last for twenty minutes or half an hour after each dose. There was no headache. That alarming symptoms may be produced by large doses, is shown by the following case. A woman, aged fifty-one, was ordered drop doses of the one per cent solution every four hours. This was taken well, and at the expiration of a week, the dose was doubled. No complaint being made, it was then increased to four minims, and after a time to six. The patient said "the medicine agreed with her," and even leading questions failed to elicit any complaint of headache or the like. After the medicine had been taken continuously for five weeks the dose was increased to ten minims. The patient then stated that the medicine no longer agreed with her; it made her sick after every dose and took her appetite away. She always vomited about five minutes after taking the medicine, the vomiting being immediately followed by headache. The medicine made her "go off in a faint" after each dose. She had three "fainting fits" in one day and could not venture to take another dose. She became quite insensible and once remained so for ten minutes. Each fainting fit was "followed by cold shivers," which "shook her violently all over." Her husband and friends were greatly alarmed, but she thought on the whole it had done her good. She had never noticed that the medicine produced drowsiness. In another case a three minim dose taken on an empty stomach caused a feeling of faintness; "everything goes dark," the patient said, "just as if I were going to faint." The patient could take the same dose after meals without the production of any unpleasant symptoms. Drowsiness is not an uncommon result of taking nitro-glycerine.

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From a consideration of the physiological action of the drug and more especially from the similarity existing between its general action and that of nitrite of amyl, I concluded that it would probably prove of service in the treatment of angina pectoris, and I am happy to say that this anticipation has been realised.

As a preliminary step I was anxious to obtain a comparative series of sphygmographic tracings. . . . Judged by the sphygmographic tracings, the effects of nitrite of amyl and of nitro-glycerine on the pulse are similar. Both drugs produce a marked state of dicrotism and both accelerate the rapidity of the heart's action. They differ, however, in the time they respectively take to produce these effects. The full action of the nitro-glycerine is not observed in the sphygmographic tracings until six or seven minutes after the dose has been taken. In the case of nitrite of amyl, the effect is obtained in from fifteen to twenty seconds after an inhalation or a dose has been taken on sugar. The influence of the nitrite of amyl is extremely transitory, a tracing taken a minute and a half after

No 1.—Before dose

No 2.—Two minutes after dose
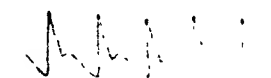
No 3.—Eight minutes after dose
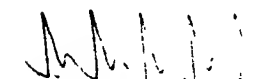
No 4.—Nine minutes after dose

No 5.—Ten minutes after dose

No 6.—Twenty two minutes after dose
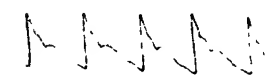
No 7.—Twenty-six minutes after dose

Influence of Nitrite of Amyl on the Pulse.

No 1.—Before inhalation
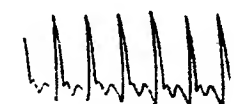
No. 2.—One minute after inhalation

No. 3.—Two minutes after inhalation.

the exhibition of the drug being perfectly normal. In fact, the full effect of the nitrite of amyl on the pulse is not maintained for more than fifteen seconds. The nitro-glycerine produces its effects much more slowly; they last longer and disappear gradually, the tracing not resuming its normal condition for nearly half an hour. The effect may be maintained for a much longer time by repeating the dose. Nitro-glycerine is more lasting in its power of producing a dicrotic form of pulse beat, and consequently in cases where the conditions of relaxation and dicrotism are desired to be maintained for a space of time, its exhibition is to be preferred to that of nitrite of amyl.

. . . .

During the last nine months I have treated three cases of undoubted angina pectoris with nitro-glycerine with what success the cases themselves will show.

William A—, aged sixty-four, first came under observation in December, 1877, complaining of intense pain in the chest, excited by the slightest exertion. It was distinctly paroxysmal, the patient being perfectly well in the intervals. The first attack was experienced in September, 1876. Patient was at the time in his usual health and was, in fact, out for a day's pleasure in the country. The pain seized him quite suddenly when walking. It was a most severe attack—as severe a one as ever he experienced in his life. It caused both him and his friends great alarm and they were most anxious that he should return home at once. He cannot tell at all what brought it on, he had been enjoying himself very quietly; it was not by any means a cold day, and he had not been running, or even walking faster than usual. He remained perfectly well until the following April when he experienced another similar attack and since then he has been suffering from them with increasing frequency. From September, 1877, they have been a source of constant anxiety and it was only by a determined effort that he could continue to follow his occupation.

The attacks usually commence with a feeling of warmth, then of heat, and then of burning heat in the chest immediately followed by a heavy pressure, from the midst of which proceeds an acute pain, so that in a moment the whole chest seems as if it were one mass of pain. It is almost impossible, he says, to describe it for he never felt anything like it before. The pain is first experienced at a small spot on either side of the sternum, corresponding to its junction with the fourth costal cartilages. From the chest the pain flies to the inner side of the arm at a point midway between the shoulder and the elbow. It runs down as far as the elbow, but never to the fingers. It is not more severe on one side than the other. During the seizure the patient suffers most acutely and feels convinced that some day he will die in an attack. He usually experiences some shortness of breath at the time, but there is no feeling of

constriction about the chest. He can speak during the seizure, though with some difficulty. The attacks are not accompanied by any sensation of warmth or chilliness, but patient is under the impression that he grows pale at the time. These attacks are induced only by exertion in some form or other, most commonly by walking, and especially by walking fast. Walking up hill is sure to bring on a seizure. Stooping down has a similar effect and the act of pulling on the boots will excite a paroxysm almost to a certainty. He is almost afraid to stoop down and when he wants to pick up anything from the floor he goes down on his hands and knees. He has a slight cough, but although it shakes him at times, it never brings on the pain. The attacks are not excited by food, but exercise taken after meals is more likely to induce them than when taken on an empty stomach. Patient has noticed that they are far more readily excited immediately after breakfast than at any other period of the day.

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There could be no possibility of doubt respecting the diagnosis. It was a typical uncomplicated case of angina pectoris.

Patient was placed for a week on infusion of quassia in order that he might be observed and also to eliminate the effects of expectation. It need hardly be said that he derived no benefit from this treatment. He was then ordered drop doses of the one per cent nitro-glycerine solution in half an ounce of water three times a day. At the expiration of a week he reported that there had been a very great improvement. The attacks had been considerably reduced in frequency and for two or three days he had had only one attack in the morning after breakfast. The attacks, when they did occur, were much less severe. He found, too, that a dose of medicine taken during an attack would cut it short. He had tried it several times, and it had always succeeded. It would not act instantly, but still very quickly; so that the attacks were considerably shortened. He was thoroughly convinced that the medicine had done him good and said he was better than he had been since first he had the attacks. . . .

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. . . Patient had adopted the plan of carrying his medicine with him in a phial and taking a dose if an attack seized him in the street. It never failed to afford relief.

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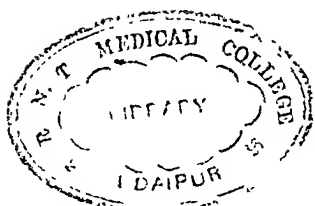
1885

PIERRE CARL ÉDOUARD POTAIN

DESCRIPTION OF THE MECHANISM OF
GALLOP RHYTHM

THE THEORY OF GALLOP RHYTHM*

By M. POTAIN

All the theories which attempted to explain the mechanism of gallop rhythm were unacceptable for clinical considerations. M. Potain undertook to establish the following theory, which he supported by graphic demonstrations.

The gallop stroke is diastolic and is due to the beginning of sudden tension in the ventricular wall as a result of the blood flow into the cavity.

It is more pronounced if the wall is not distensible and the failure of distensibility may depend either on a sclerotic thickening of the heart wall (hypertrophy due to Bright's disease) or to decrease in muscular tonicity. Since the wall, by virtue of its own elasticity, is no longer able to resist the inflow of blood, it is placed under tension precisely at the same moment that this occurs.

The gallop can originate in all cases where the elastic resistance of the wall encroaches on muscular tonicity, either by an increase of the first factor, or a diminution of the second.

It was observed in a goodly number of acute diseases, chiefly in typhoid fever, and also in cachectic subjects, in whom cardiac function is embarrassed. It occurs either in a constant or more pronounced manner in ventricular hypertrophy of Bright's disease, or on the other hand, accompanies dilatation of the right heart of hepato-gastro-intestinal origin. It is therefore an important sign owing to the latent or insidious character of the latter diseases.

The name, gallop rhythm, was first introduced by Bouillaud† and should be used for the phenomenon to which it applies; the phenomenon does not, however, always maintain the character of the gallop of a horse.

One could apply the name of murmur of diastolic shock in all the cases in which the above mentioned theory is applicable and reserve the term, gallop rhythm, specifically for the type, in which the *anapest* rhythm occurs.
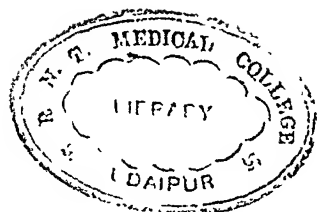
1887

AUGUSTUS D. WALLER

DEMONSTRATION OF A METHOD OF LEADING OFF
THE ACTION CURRENTS OF THE HEART BY
MEANS OF CONTACT ELECTRODES

AUGUSTUS DÉSIRÉ WALLER

(1856-1922)

*A*UGUSTUS DÉSIRÉ WALLER was born in Paris on July 12, 1856. He was the only child of Dr. Augustus Volney Waller, a physiologist of international reputation. Dr. Waller, Senior, had observed the emigration of leucocytes in 1846, and had described the ciliospinal region in 1851. In 1853 he had emphasized the vasoconstrictor action of the sympathetic nerves, and in 1856 he had observed that nerves, when separated from their cells of origin, undergo degeneration.

Young Waller received his academic training at the Collège de Genève. On the death of his father in 1870, the widow and son moved to Aberdeen, Scotland. In due time Waller matriculated at the University of Aberdeen, where he began the study of medicine. At Aberdeen in 1878 he received the medical degrees, Bachelor of Medicine and Master of Surgery. He continued his medical training at the University of Edinburgh, where, according to the editor of the "Lancet," he received the degree of Doctor of Medicine in 1881.

Soon after graduation, Waller went to London, where he worked under Burdon-Sanderson in his physiologic laboratory. In 1880 and again in 1883 he received scientific grants from the British Medical Association. In 1884, he was appointed a research scholar. About this time he received the appointment of lecturer on physiology at the London School of Medicine for Women. There he met, and afterwards married, Alice Mary, the second daughter of George Palmer of Reading, a member of Parliament.

Soon after his marriage, Waller was appointed lecturer on physiology in the Medical School of St. Mary's Hospital, where he taught for sixteen years. In 1902, the Senate of the University of London established a physiological laboratory and Waller was appointed its director. There he continued a brilliant career until his death in 1922. He died at his home in St. John's Wood on March 11, 1922, as the result of a cerebral hemorrhage.

Waller's most important contributions to medicine were in the field of electrophysiology. He was the first to demonstrate that the currents set up by the beating of the heart in animals could be recorded without opening the thorax. He was the first, also, to obtain an electrocardiogram of the action of the human heart. We are reprinting his first classic contribution in this field, a demonstration of leading off the action currents of the heart by means of contact electrodes. This paper appeared in the "Journal of Physiology" for 1887.

A summary of Waller's electrophysiologic work may be found in his Oliver-Sharpey Lectures on the electrical action of the human heart, delivered before the Royal College of Physicians in 1913.

In the early part of his career, Waller was much interested in investigating the electric currents in living structures, especially in nerve and muscle, but also in the skin, the retina, and in plants. In 1903 he summarized these observations in his book, "Signs of Life from their Electrical Aspect."

Waller is also remembered for his observations on the effects of gases and anesthetic vapors on the irritability of nerves and muscles. Having been a fellow of

the Royal Society since 1892, he was asked, in 1896, to deliver the Croonian Lecture before that society. In this lecture he expressed his ideas concerning his studies of isolated nerves. In 1897, as president of the Section on Anatomy and Physiology of the British Medical Association, he read a paper on the relative efficacy of ether and chloroform as anesthetics.

During his last years, Waller was primarily interested in two subjects of importance, the physiologic cost of muscular work as measured by the amount of carbon dioxide exhaled, and the emotive response of man to pain or the threat of pain.

Waller was the author of an important textbook, the "Introduction to Human Physiology," first published in 1891 and later in several other editions. He also contributed several articles to the "Transactions" of the Royal Society and to the "Journal of Physiology." In 1909 he delivered the Hitchcock Lectures at the University of California.

For his outstanding scientific contributions Waller received, besides many national honors, membership in many scientific societies abroad. He was a corresponding member of the Société de Biologie, Paris, a member of the Physiological Society of Moscow, and of the Royal Academies of Medicine of Rome and Brussels. For his electrophysiologic work he was elected a lauréat de l'Institut de France.
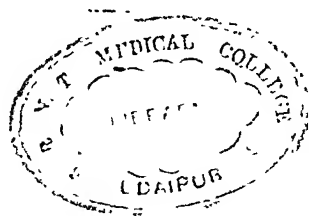
its visible movement, and the current of action of the heart begins before the commencement of the heart's contraction. For muscle, the time-difference given is $\frac{1}{200}''$, for the heart (rabbit) $\frac{1}{10}''$; for the frog's heart, the rheotome observations of Marchand[1] are to the effect that the variation begins 0.01″ to 0.04″ after excitation, while the contraction does not begin until 0.11″ to 0.33″. The capillary electrometer may with advantage be employed to measure this time-difference, the electrical and the mechanical events being simultaneously recorded. This I carried
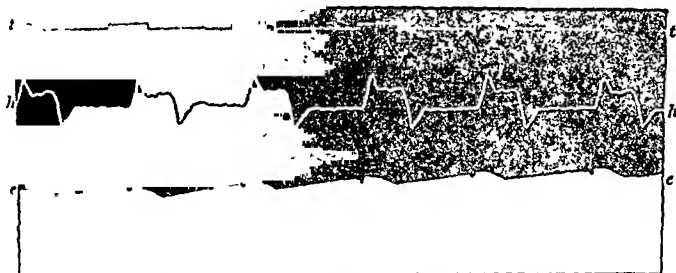
Fig. 1. Man. Heart led off to electrometer from front and back of chest (front to Hg; back to H_2SO_4).

e.e. electrometer.　　h.h. cardiograph.　　t.t. time in seconds.
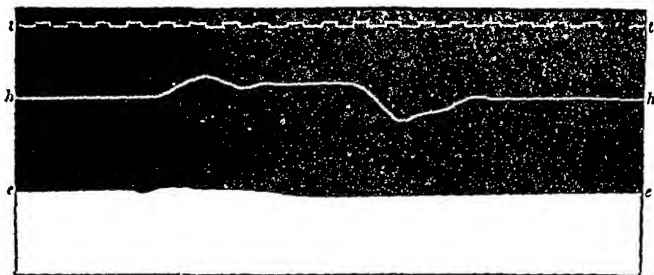
Fig. 2. Man. Heart led off to electrometer from front and back of chest (front to Hg; back to H_2SO_4).

e.e. electrometer.　　h.h. cardiograph.　　t.t. time in $\frac{1}{10}$th sec.

out on voluntary and upon cardiac muscle with the same instrument as that which I employed for the human heart, and thus ascertained that its indications are trustworthy in this capacity.

In all these cases the antecedence of the electrical variation is clear and measurable. In the case of the excised kitten's heart, the time-difference is about 0.05″ with a length of contraction of about 2″, i.e., the interval between the electrical and the mechanical event is increased in the sluggishly acting organ. In the case of the human heart, the time-difference appears to be about 0.015″ with a length of systole of 0.35″—

[1]Pflüger's Archiv XV, 1877, p. 511.

a value which corresponds with that obtained by Donders for the rabbit's heart in situ by the method of the secondary contraction, viz. $\frac{1}{10}''$ (the length of systole being presumably about $\frac{1}{3}''$).

That a true electrical variation of the human heart is demonstrable may further be proved beyond doubt by leading off from the body otherwise than from the chest wall. If the two hands or one hand and one
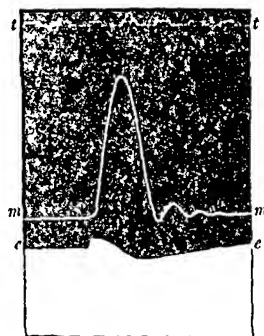
Fig. 3. Frog. Gastrocnemius led off to electrometer from the middle of the muscle and from the tendon. Contraction excited by a single break induction shock applied to the sciatic nerve.

e.e. electrometer.   *m.m.* muscle.   *t.t.* time in $\frac{1}{10}$th sec.

(muscle to H_2SO_4; tendon to Hg).

The diphasic variation (1st phase middle negative to end; 2nd phase end negative to middle) begins about ·01″ before the commencement of muscular contraction.
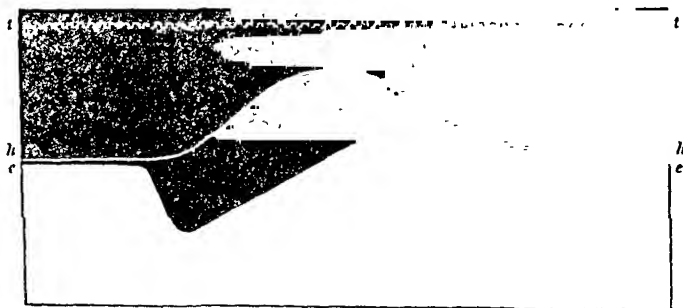
Fig. 4. Frog's heart. Spontaneous contraction.

e.e. electrometer.   *h.h.* heart's contraction.   *t.t.* time in $\frac{1}{10}$th sec.

(apex to H_2SO_4, base to Hg).

The variation is diphasic—S. N.

The first phase begins $\frac{1}{10}''$ before the commencement of contraction.

foot be plunged into two dishes of salt solution connected with the two sides of the electrometer, the column of mercury will be seen to move at each beat of the heart, though less than when electrodes are strapped to the chest. The hand and foot act in this case as leading off electrodes from the heart and by taking simultaneous records of these movements of

the mercury and of the movements of the heart it is seen that the former corresponds with the latter, slightly preceding them and not succeeding them as would be the case if they depended upon pulsation in the hand or foot. This is unquestionable proof that the variation is physiological, for there is here of course no possibility of altered contact at the chest wall, and any mechanical alteration by arterial pulsation could only produce an effect 0.15″ to 0.20″ after the cardiac impulse. A similar result is obtained if an electrode be placed in the mouth while one of the extremities serves as the other leading off electrode. The electrical variation precedes the heart's beat as in the other cases mentioned.

In conclusion it will be well to allude to the difficulties which arise in the interpretation of the character of the electrical variation of the human heart.
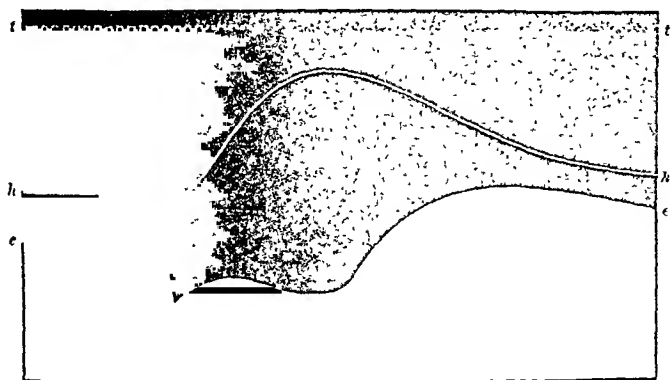
FIG. 5. Kitten's heart, excised.
(apex to Hg. base to H₂SO₄).
e.e. electrometer.   *h.h.* cardiograph.   *t.t.* time in 1/10th sec.

By mere inspection of the electrometer, it is often most difficult to determine the direction of very rapid movements of the mercury, and photography must be employed. But even then, owing to the small amplitude of the movement, it is still difficult to say whether the variation consists of two movements, and whether each movement indicates a single or a double variation in the same direction. Differences in the position of the electrodes also give rise to differences of the apparent variation. Thus with the following position of the electrodes (Hg electrode over the apex beat, H_2SO_4 electrode on the right side of the back) the variation as watched through the microscope appears usually nN, and changes to SN if the Hg electrode be shifted to the sternum. If the Hg electrode is on the back and the H_2SO_4 electrode over the apex beat, the variation appears to be sS and to become nS when the H_2SO_4 electrode is shifted away from the apex beat. The variations accompany-

ing the heart's beat observed as carefully as possible (without the aid of photography) on a healthy person with different positions of the leading off electrodes were as follows. It is to be remarked that the direction of variation as observed in this series is not such as to indicate negativity of the cardiac electrode but the reverse.

						Electrodes reversed
Precordium	to H_2SO_4	Back	to Hg	SS variation		NN
"	"	Left hand	"	SS	"	NN
"	"	Right hand	"	SS	"	NN
"	"	Left foot	"	SS	"	NN
"	"	Right foot	"	SS	"	NN
Left hand	"	Right hand	"	SS*	"	NN
"	"	Left foot	"	?	"	?
"	"	Right foot	"	?	"	?
Right hand	"	Right foot	"	NN*	"	SS
"	"	Left foot	"	NN	"	SS
Right foot	"	Left foot	"	0	"	0
Mouth	"	Precordium	"	NN	"	SS
"	"	Right hand	"	0	"	SS
"	"	Left hand	"	NN*	"	SS
"	"	Right foot	"	NN	"	SS
"	"	Left foot	"	NN	"	SS
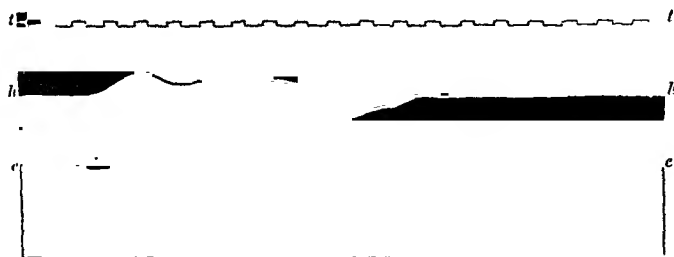
Fig. 6. Man. Led off to electrometer by right hand and right foot.
(hand to Hg. foot to H_2SO_4).

c.c. electrometer. *h.h.* cardiograph. *t.t.* time in $\frac{1}{26}$th sec.

Fig. 7. Man. Led off to electrometer by mouth and left foot.
(mouth to Hg. foot to H_2SO_4).

e.e. electrometer. *h.h.* cardiograph. *t.t.* time in $\frac{1}{26}$th sec.

It is on account of these sources of doubt that I have not thought it advisable at this stage to attempt a definite interpretation of the character of the variation, which although as shown, especially by the experiments illustrated in Figs. 6 and 7, is certainly physiological, may nevertheless be physically complicated by the conditions of demonstration on the human body.
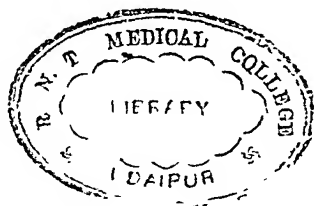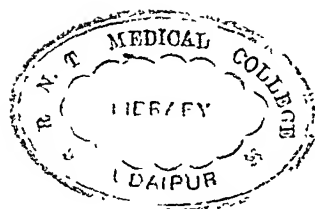
1887

JOHN A. MacWILLIAM

THE EXPERIMENTAL PRODUCTION OF EXTRASYSTOLES,
VENTRICULAR FIBRILLATION AND
AURICULAR FLUTTER

JOHN A. MacWILLIAM
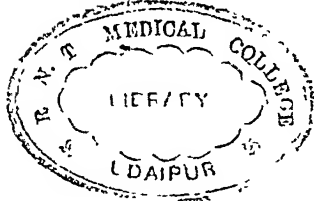
JOHN ALEXANDER MacWILLIAM
(1857-1937)

*J*OHN ALEXANDER MacWILLIAM was born in 1857 in Culmill, Scotland. He studied medicine at the University of Aberdeen, where he received the degrees of Bachelor of Medicine and Master of Surgery in 1880. For his high scholastic standing he was awarded the John Murray Medal. In 1882 he received the degree of Doctor of Medicine with honors. His thesis concerned the cardiac muscular fiber in the various classes of the animal kingdom, and the diaphragmatic fiber in various animals.

MacWilliam spent some time in postgraduate work at the Universities of Edinburgh and Leipzig, and at University College in London. At the University of Leipzig it was his good fortune to work under Karl Ludwig. There MacWilliam, with Henry Pickering Bowditch (1840-1911) of Harvard, and Walter Holbrook Gaskell (1847-1914) of Cambridge, studied the physiologic properties of heart muscle.

In 1882, MacWilliam was appointed demonstrator of physiology at University College in London. He held this position until 1886, when he was appointed professor of physiology at the University of Aberdeen. In London, he had also served for some time on the staff of the Charing Cross Hospital Medical School and lectured on physiology at the London School of Medicine for Women.

When MacWilliam had been at the University of Aberdeen for not quite a year he published his important paper, "Fibrillar Contraction of the Heart" (1887). This paper we are reproducing for our readers. It contains his discovery that fibrillar contraction of the heart is caused by a lack of harmony in the contraction and relaxation of the minute muscular fibers which compose the walls of the heart. He showed that fibrillation is brought about by a "rapid succession of incoordinated peristaltic contractions." He also described the relationship of the refractory period to this disturbance, and he gave evidence that certain poisons, when injected into the blood stream, led to the occurrence of fibrillar contractions of the ventricles. According to an editorial in the "Lancet," MacWilliam, as early as 1887, believed that sudden death, during the administration of chloroform, is the result of ventricular fibrillation.

MacWilliam contributed many articles to medical literature. In 1913 he wrote an important paper for "Heart," entitled, "Blood Pressures in Man in Normal and Pathologic Conditions." Later, in 1925, he again contributed an article on this subject to the "Physiological Review."

In 1916, MacWilliam was elected a fellow of the Royal Society. Many of his papers are contained in the "Proceedings" of this Society, including his studies on the action of chloroform, and his studies on ether, proteins, and muscle sounds.

He retired from his professorship in 1927 to become emeritus professor, and in 1928 the University of Aberdeen conferred upon him the honorary degree of Doctor of Laws.

He died on January 13, 1937, in his eightieth year. He was married twice and is survived by his widow.

FIBRILLAR CONTRACTION OF THE HEART*

By

JOHN A. MacWILLIAM, M.D.

Professor of the Institutes of Medicine in the University of Aberdeen

MANY years ago Ludwig and Hoffa[1] showed that the application of strong constant currents or faradic currents to the ventricles of the dog's heart causes an abolition of the normal beat. The ventricular muscle is thrown into a state of irregular arhythmic contraction, whilst there is a great fall in the arterial blood pressure. The ventricles become dilated with blood as the rapid quivering movement of their walls is insufficient to expel their contents; the muscular action partakes of the nature of a rapid inco-ordinated twitching of the muscular tissue. This condition persists for a very long time in the dog, and as Ludwig showed, it is possible to kill an animal in this way—by applying a faradic current to the ventricles. The auricles go on beating rhythmically; they do not participate in the irregular movement excited in the ventricles. These phenomena are familiar to all who have worked much with the mammalian heart; they have been designated by various names—Herzdelirium, Delirium cordis, Fibrillar contraction, Intervermiform movements, etc.

During the last two years I have performed a large number of experiments bearing upon this subject. My earlier investigations were pursued in the physiological laboratory of University College, London, and the more recent ones in the physiological laboratory of the University of Aberdeen. I have studied the phenomena in question in the hearts of the dog, cat, rabbit, rat, mouse, hedgehog, and fowl: both in the young animal and in the adult.

The experiments were all conducted on completely anaesthetised animals, artificial respiration was carried on, a cannula being inserted in the trachea; the thorax was opened in many cases and the heart laid bare; the temperature of the animal was kept up by means of a warm pan.

I shall briefly state the main facts in my investigation.

I. The state of arhythmic fibrillar contraction is essentially due to certain changes occurring within the ventricles themselves. It is not due to the passage of any abnormal nerve impulses to the ventricles from other parts, or to the interruption of any impulses normally transmitted

to the ventricles and necessary for their normal co-ordinated action. The condition is not due to injury or irritation of the nerves that pass over the ventricles from the base of the heart.

The ventricles contain within themselves the entire mechanism necessary for the execution of regular co-ordinated beats. They are not dependent for this power on any nervous or mechanical connection with other parts. The continuity of the nerves that pass from the auricles to the ventricles is not at all essential for the execution of regular and effective beats by the ventricles; nor is the mechanical connection between those parts necessary. This is obvious from the fact that when a section is made through the auriculo-ventricular groove so as to separate the ventricles entirely from the auricles, the isolated ventricles can still exhibit their co-ordinated rhythmic contraction. Instead of cutting off the ventricles, Wooldrige[1] and Tigerstedt[2] physiologically disconnected the ventricles from the auricles so as to destroy all vital connection between them while the parts were still kept *in situ* and the flood of blood through the cavities of the heart was allowed to go on; the ventricles went on beating in regular fashion though at a slower rate than before. I have frequently performed a similar experiment and have watched the ventricular action as it went on, strong and regular for prolonged periods. It is evident that neither the nervous, nor the mechanical connection between the auricles and the ventricles is necessary for the effective contraction of the latter. It is clear that a mere solution of the continuity of the nerves passing to the ventricles does not destroy the character of the ventricular beat; and it is plain, that such a solution of continuity cannot be the cause of a sudden replacement of the normal systole by the arhythmic fibrillar form of contraction.

Nor is the fibrillar contraction due to irritation of those ventricular nerve trunks. Many observers have noticed its occurrence when the nerve trunks on the surface of the ventricles were being stimulated. But such results appear to be due entirely to an escape of the exciting current to the underlying ventricular substance. For when the nerve trunk is isolated for some little distance and precautions are taken to prevent an escape of the current, I have never found the nerve stimulation to have any effect at all in inducing the fibrillar contraction. Moreover, an interrupted current readily brings about the arhythmic fibrillar condition when applied to regions of the ventricles where there are no nerve trunks, e.g., to the very apex of the heart. Even mechanical or thermal stimulation applied to this region may lead to the same result.

The arhythmic fibrillar contraction is undoubtedly a phenomenon depending on changes within the ventricular substance; it can occur quite independently of any mechanical relation of the ventricles to the rest of the heart, and of any nervous relation of the ventricles to the rest of the

[1] Arch. f. Anat. u. Physiol., 1883.
[2] Arch. f. Anat. u. Physiol., 1884.

heart or to the extra-cardiac nerves. The isolated ventricles whether in the quiescent state or beating rhythmically, can by the application of faradic currents be readily thrown into the characteristic fibrillar state, just like the ventricles of an intact heart. And in the intact heart the fibrillar contraction appears to be entirely uninfluenced by nerve excitation of any kind; stimulation of the vagus or any other nerve appears to produce no effect whatever.

Further, the fibrillar contraction can be propagated from one part of the ventricular substance to another quite independently of the nerve trunks. For if a number of overlapping incisions be made across the long diameter of the ventricles so as to leave the apex attached to the rest of the ventricles by a zig-zag isthmus of tissue, it often occurs that fibrillar movement excited by faradisation in the apex travels along the zig-zag isthmus of connecting substance, and so comes to pervade the whole of the ventricular tissue.

II. The arhythmic fibrillar contraction is not necessarily dependent on the destruction or paralysis of a co-ordinating centre located in any particular part of the ventricles.

Kronecker and Schmey[1] succeeded in throwing the ventricles of the dog's heart into the state of fibrillar movement by piercing with a needle a certain limited part of the ventricular septum near the junction of its upper and middle thirds. This result these investigators attributed to the destruction of a centre located in that region, and normally presiding over the co-ordination of the ventricular muscle in the execution of its regular beat.

There is conclusive evidence that all cases of fibrillar contraction of the ventricles cannot be explained by such a hypothesis—the destruction of a co-ordinating centre localised as indicated above. The fact that recovery may take place—that the ventricles may resume their co-ordinated rhythm, controverts the idea of the actual destruction of a centre essential for co-ordination. Such recovery I have witnessed in several instances in the dog's heart, and in a very large number of instances in the hearts of other animals (cat, rabbit, rat, mouse, hedgehog, and fowl). Recovery occurs with different degrees of facility in different animals and in different conditions in the same animal. In the dog, recovery occurs with much difficulty and only after the fibrillar contraction has lasted for a considerable space of time; indeed there very frequently is no recovery apparent—the ventricles may not recommence beating after the inco-ordinated quivering movement has ceased. At times, however, a number of regular beats are seen after the termination of the fibrillar contraction. A depression of the excitability of the ventricular tissue often appears to favour recovery.

[1] Sitzungsber. d. Berliner Acad. 1884.

In most mammals, recovery commonly occurs. Very often it is possible to induce the fibrillar movement again and again, complete recovery occurring in the intervals, when the normal systoles are seen. In young mammals, foetal or after birth, recovery appears to be the rule; the fibrillar movement is only a temporary condition, and soon gives place to normal beats.

In birds also I have frequently observed complete recovery. The fibrillar condition is readily induced by faradisation. The ventricles exhibit the characteristic quivering movement; they become dilated with blood. In consequence of the stagnation of blood in the ventricles, the auricles also become gorged and may become so over-distended that they temporarily stop beating; asphyxial convulsions occur in the skeletal muscles. After a time, however, the fibrillar movement ceases, the ventricles remain quiescent for a little time, then give a regular co-ordinated beat and the action of the whole heart proceeds in the normal fashion. These phenomena can by the application of a current of the proper strength be induced again and again.

Further, in addition to the evidence afforded by the recovery of the ventricular beat, there is the fact that the arhythmic fibrillar movement may very readily be induced by means that are not capable of destroying a deep-seated co-ordinating centre, e.g., faradic, mechanical, or thermal stimulation of the surface of the ventricles even at the very apex.

Since it is certain that the arhythmic fibrillar movement is not necessarily due to the actual destruction of a co-ordinating centre, there next arises the question as to whether the fibrillar contraction may be due to the temporary paralysis of such a centre as that indicated by Kronecker—of the existence of which no histological evidence has, as far as I am aware, been advanced.

I shall at a later stage of this paper have to adduce some evidence regarding the action of certain poisons which when injected into the blood lead to the occurrence of fibrillar contraction of the ventricles. Such a result might be regarded as due to the paralysis of a hypothetical co-ordinating centre. And the fibrillar contraction caused by stimulation (electrical, mechanical, etc.) of the ventricular surface might be explained in a somewhat similar fashion. For it is conceivable that such stimulation might give rise to strong abnormal afferent impulses with the result of deranging or paralysing the action of the co-ordinating centre; the paralysis might be a temporary one or might be permanent according to the particular circumstances in each case.

But there is strong evidence against the adoption of such a view—against the idea that the phenomena are due to the behaviour of a definite co-ordinating centre localised above the middle of the ventricular septum in the dog's heart. For the influence of such a centre does not appear to be at all essential for the production of co-ordinated and efficient beats.

The amputated apex—the lower third or fourth of the ventricles—both in the dog and in all other mammals I have examined,—is capable of executing co-ordinated beats when it is entirely removed from all possible relation with any co-ordinating centre high up in the ventricular septum. This one can verify by the rough but conclusive experiment of tying the freshly removed apex of a vigorous heart upon a double cannula through which the cavity of the left ventricle can be filled with blood; the propulsion of fluid at each beat of the isolated apex can be readily observed. The visible character of the beat may also be noted, and the co-ordinated nature of the contraction causing a marked diminution of the cavity at each systole may be felt with the finger tip inserted into the cavity of the left ventricle. It is obvious then that the paralysis of a co-ordinating centre in the upper half of the ventricular septum would not necessarily cause a loss of co-ordination in the contraction of the whole of the ventricular muscle.

Further there is the fact that the apical portion of the ventricles—capable as it is of performing regular beats—can be thrown into a state of fibrillar contraction by the usual means, e.g., the application of a faradic current. In the isolated apical part of the ventricles (in all the mammals I have examined) I have been able to excite the fibrillar contraction again and again, recovery occurring in the intervals, and co-ordinated beats being given in response to single stimuli applied during those intervals. It appears then that the behaviour of the intact ventricles and of the entire isolated ventricles both as regards co-ordinated single beats and as regards the fibrillar contraction can be reproduced in the isolated apical portion; and hence we may conclude that these phenomena are not necessarily dependent on the condition of any co-ordinating centre in the upper half of the ventricles.

III. The outstanding features of the arhythmic fibrillar contraction are: (1) the complexity of movement, (2) its persistence, (3) its rapidity.

The complexity of the fibrillar movement appears to be in direct relation to the complex arrangement of the muscular fibres of the ventricular walls.

In the ventricles we have bundles of muscular fibres forming by their interlacement a texture of remarkable complexity. It appears that the complex quivering movement depends on the passage of rapidly repeated waves of contraction along the complexly arranged muscular bundles which are enclosed by connective tissue and joined to one another by cross-branches. It is readily conceivable that contractions simply conducted along the muscular fibres should be transmitted with unequal rapidity along the ventricular walls and should reach the same part of the ventricular wall at different points of time. Some bundles of fibres are in a state of contraction while neighbouring bundles are relaxed

and so instead of a co-ordinated contraction causing a definite and (in the case of the left ventricle) concentric narrowing of the ventricular cavity, there occurs an irregular and complicated arhythmic oscillation of the ventricular walls which remain in a position of diastole.

That the complexity of the fibrillar movement in the grown animal depends on the character of the muscular structure is illustrated by the appearances presented by the corresponding movements in the hearts of foetal and young animals. In these as long as the structure of the ventricles is simple the rapid movement excited by faradisation is of a simple character. And just as the complexity of the muscular structure increases in the growing animal so does the complexity of the movement obtained. There can be observed a complete gradation from the simple movement excited by faradisation in the ventricles of the mammalian foetus or of the chick (a movement much resembling that seen in similar circumstances in the comparatively simple ventricles of cold-blooded animals) to the very characteristic and striking complexity of the fibrillar contraction in the adult mammal or bird. It is obvious that the nature of the muscular structure is a cardinal feature, and it is not very evident why such should be the case if the condition is due to derangement of a nervous mechanism causing it to discharge irregularly; for a deranged nervous mechanism discharging irregularly might cause an equally irregular movement whether the muscular arrangement is simple or complex.

The simpler character of the movement excited by faradisation in the auricles of warm-blooded animals is probably due to the simpler histological structure of the auricular walls and the simpler mode of propagation of the normal contraction.

The persistence of the fibrillar contraction appears to depend on the high excitability of the ventricular tissue.

When the fibrillar contraction has been brought about by stimulation of the ventricles, the prolonged continuance of the movement, after the cessation of the exciting cause is a striking feature. It appears to be a result of the excitation of a highly excitable, and probably highly rhythmic tissue. The duration of the movement varies in each instance with the excitability of the ventricular muscle. It can easily be shown, that in certain depressed conditions of the ventricular tissue, the duration of the fibrillar movement, induced by stimulation, is much diminished, and when the ventricular excitability is very much lowered, (by gradual cooling, exhaustion, etc.) it frequently occurs that the fibrillar contraction does not persist after the stimulating current is discontinued; it simply occurs during the passage of faradic current and passes off at the cessation of that current. Indeed, in some instances it may be found that the fibrillar contraction cannot be excited at all by faradisation, whilst the ventricles are still capable of executing single beats. A cer-

tain degree of excitability is necessary for the production of the fibrillar contraction in response to stimulation.

Similar facts with reference to the duration of movement, after the discontinuance of the exciting cause, may be seen in the hearts of cold-blooded animals. In the heart of the eel, for example, where there are a number of parts possessed of different degrees of excitability and rhythmic power, very marked differences are to be observed in the behaviour of the several parts after a stimulating current has been temporarily applied. The sinus with the basal wall, and the canalis auricularis, the auricle, and the ventricle, form a descending series as far as rhythmic power is concerned and they present similar differences as regards the after effects of stimulation. In the ventricle a short period of moderate stimulation excites a movement, which usually terminates immediately or very soon after the end of the stimulation; the precise period at which the movement terminates varies according to the strength of the exciting current and the excitability of the ventricle; in a very excitable ventricle (in situ, with the normal circulation intact) the movement may persist for some little time after the stimulation has ended. In the auricle, the movement usually persists longer, and in the sinus a great deal longer still. Indeed, in the sinus a single stimulation can often lead to a series of beats, whereas in the case of the auricle and still more in the ventricle a single stimulation excites but a single contraction. Moderate heating of the tissue causing a rise in its excitability usually leads to a marked increase in the persistence of the movement excited by a short period of stimulation.

Similarly in the mammalian heart the duration of the fibrillar movement after the end of the period of excitation varies. In the foetal heart it lasts but a short time, and in adult hearts that have been much depressed by exhaustion and by gradual cooling, the fibrillar movement usually passes away very much earlier than it does in a more excitable heart.

The mechanism of the movement, as will be subsequently stated, appears to be such as to involve its continuance as long as the excitability of the ventricular tissue is sufficiently high.

The cause of the great rapidity of the series of contractions that course over the ventricular fibres during the state of fibrillar contraction will be considered later on.

IV. The arhythmic fibrillar contraction is in one class of cases a phenomenon of irritation induced by the action of various recognised stimulants.

The state of excitement generated in the muscular tissue appears to resemble in some respects the state of excitement obtaining in the nerve cells of the cortex cerebri during an attack of epileptiform convulsions induced by strong stimulation.

It has been stated that the duration of the fibrillar contraction depends on the excitability of the ventricular tissue. In like manner the readiness with which the fibrillar contraction can be excited by stimulation, is in close relation with the ventricular irritability. In a depressed heart it is frequently very difficult to produce the phenomenon in question by stimulation; very powerful currents are necessary.

On the other hand, when the excitability is heightened, it is easy to induce the fibrillar contraction. The occurrence of this phenomenon in response to stimulation is retarded and its duration shortened by conditions that depress the excitability of the cardiac muscle; its occurrence is favoured and its duration prolonged by causes that augment the cardiac irritability. In an exhausted heart it can frequently be seen that faradisation of the right ventricle leads to the occurrence of the fibrillar contractions in both ventricles, when such a result has ceased to be obtained by faradisation of the left ventricle. The difference in the behaviour of the ventricles, in this respect appears to be due to the greater persistence of the excitability in the right ventricle as compared with the left.

When the fibrillar contraction has been excited by stimulation it can often be arrested by the cautious application of depressant measures calculated to diminish the excitability of the ventricular tissue, e.g., deprivation of blood supply and cooling.

The readiness with which the ventricles are thrown into the fibrillar condition varies remarkably in different conditions of the cardiac tissues. In a normally contracting and vigorous heart it usually requires a faradic current of considerable strength to produce the result in question. And it is not easy in these circumstances to induce the fibrillar contraction by mechanical or thermal stimulation. But in certain changed conditions of the organ it becomes extremely easy to throw the ventricles into the fibrillar movement. An exceedingly weak faradic current, a touch with a hot wire, a mere scratch with the point of a pin, slight friction of the ventricles against the cut end of a rib, or even slight pressure with the finger are each of them sufficient at such times to excite the fibrillar contraction. The precise conditions in which there is such a remarkable sensitiveness to certain forms of stimulation are difficult to define; I have frequently observed such a sensitiveness when the action of the heart has been deranged or impaired by various causes—among others by a temporary arrest of the respiration or by a great fall in the blood pressure leading to anaemia of the cardiac tissues, et cetera; the phase of increased sensitiveness seems to be a transitory one.

The frequent occurrence in the ventricles of such phases of extreme readiness to assume the fibrillar form of contraction appears to me to be of great importance with regard to the question of electrical stimulation of the heart in man during sudden cardiac failure (syncope during

the administration of anaesthetics, etc.). It is obvious that the use of faradic currents of any strength is attended with grave danger in such cases. For although Von Ziemssen and others have applied the induced current to the human heart without any serious results, the conditions were different in such cases. They experimented with normally beating hearts, the tendency of which to assume the fibrillar form of contraction is strikingly less than what frequently obtains in hearts placed in abnormal circumstances—necessarily present in those cases where the faradic current is employed clinically.

But although the exposed heart in the opened thorax may be readily thrown into the arhythmic fibrillar contraction by faradisation, it may be urged that possibly the normally beating heart in the intact thorax, is not similarly affected. I have on several occasions introduced a fine platinum wire electrode through the chest wall so as to come in contact with the ventricles and have then faradised, the other electrode being applied to the outside of the chest wall; the fibrillar contraction was at once induced.

By the use of single induction shocks I have never seen the fibrillar contraction excited either when the shock is passed through the thoracic walls or when it is applied to the exposed heart. The single induction shock seems to be free from the dangers accompanying the use of the faradic current. Hence I have urged its superiority as a means of cardiac stimulation, in a paper to be read at the Ninth International Medical Congress at Washington.

The extreme readiness with which in certain circumstances the ventricles are thrown into the fibrillar contraction by any form of irritation, mechanical as well as electrical, renders it apparent that the experiment of puncturing the heart in order to destroy a certain part is attended with many difficulties. For very frequently the mere mechanical irritation would be amply sufficient to produce all the phenomena usually resulting from faradisation. And this condition of increased sensitiveness to irritation and increased tendency to assume the fibrillar mode of contraction appears to occur with special frequency and to a very marked degree in the heart of the dog.

V. In another class of cases the fibrillar contraction is induced by the more or less sudden action of certain influences of a depressing nature.

The injection of certain salts (e.g., bromide of potassium in strong solution) into the blood appears to induce the fibrillar condition in a very short space of time (frequently within one minute). A dose of about 0.1 gramme is sufficient in the hedgehog.

When such an injection is made (cat and hedgehog) there is almost immediately a marked change in the character of the systole. The origin and course of the contraction become very apparent both in the auricles and in the ventricles. In the former it passes forwards from the entrance

of the great veins; in the latter it sweeps from the base of the heart towards the apex; on the front of the heart the contraction can be most distinctly seen beginning at the conus arteriosus and passing downwards. The ventricles become dilated with blood; the contractions are evidently unable to empty the cavities. When the heart is in a depressed state no further important change may be observed; the contractions gradually become weaker and slower until they cease altogether. But in the case of a vigorous heart there usually occurs a striking change—a short time after the injection of the bromide. The ventricles go into the state of fibrillar contraction with its usual features.

I have not as yet seen any complete recovery from the inco-ordinated condition produced in this way. The ventricles do not seem to recover their power of giving regular beats. Single contractions may occur after the rapid quivering movement has ceased but they appear to be fibrillar in their nature. And any contractions excited by single induction shocks in such circumstances appear to be of the same character.

After the injection of a solution of atropin I have observed somewhat similar phenomena; here, however, the fibrillar movement was arrested by the injection of pilocarpin and complete recovery of the ventricular beat took place.

I have on some occasions observed phenomena of the same kind when an animal (cat) was suddenly and powerfully cooled by the application of a mixture of ice and salt to the surface of the skin and the insertion of an ice bag into the abdominal cavity. After the cooling had gone on for a time, the ventricles suddenly passed into the state of fibrillar contraction.

See and others have described the occurrence of a similar fibrillar movement in the dog's ventricles as one of the results of sudden occlusion of the coronary arteries.

VI. The arhythmic fibrillar contraction is fundamentally different from a rapid series of normal contractions. Its genesis probably assumes in all cases one or other of two forms.

It is probable that the normally contracting ventricles possess within themselves certain co-ordinating arrangements in virtue of which the muscular contraction constituting a normal beat rapidly traverses the whole of the ventricular substances, causing a uniform or nearly uniform contraction of all the fibres of the ventricular walls thus leading to a concentric narrowing of the ventricular cavity and a consequent expulsion of its contents. The co-ordinating arrangements appear to exist in the lower portion of the ventricles as well as in the upper portion; for it has been seen that the apical part can execute co-ordinated beats when severed from the rest of the heart.

A normal co-ordinated contraction appears to be essentially different from the individual beats that may be seen after poisoning with bromide

of potassium and occasionally in other conditions. In the latter case the contraction is obviously of a peristaltic nature; the contraction wave can be seen passing over the ventricular surface in definite directions. The contraction may be caused to start at any part in the ventricular substance by the application of a single direct stimulus; the contraction begins in the stimulated area and hence spreads over the rest of the ventricles; a phenomenon precisely similar to what one sees in the hearts of cold-blooded animals.

The peristaltic contraction evidently passes over the various interlacing bundles at different points of time, so that the whole thickness of the ventricular wall at any part is never uniformly contracted. Hence there is a wiry feel distinctly perceptible when the ventricles are held between the fingers as the peristaltic contraction is passing through its substance; certain fibres are hardened by the presence of contraction in them while neighbouring fibres are relaxed and soft. Such peristaltic contraction appears to be incapable of emptying the ventricular cavities of their contents; it appears to be essentially different from a co-ordinated beat however slow the latter may be. A co-ordinated beat never presents a wiry feel to the finger; it gives the sensation of a steady and uniform hardening of the muscle substance—of precisely the same nature as the hardening one feels in a skeletal muscle during its contraction. The contraction seems to involve as a whole the complicated interlacement of fibres forming the ventricular wall.

It appears then that the ventricles are capable of executing two forms of beat. One is the co-ordinated contraction seen in the normal heart and capable of being excited by artificial stimulation (e.g., by single induction shocks) either in an intact heart, or in the fresh and vigorous excised ventricles or ventricle-apex. The other form of beat is the inco-ordinated or simple peristaltic contraction, such as may be seen after poisoning with bromide of potassium and in certain other conditions.

VII. The state of arhythmic fibrillar contraction (delirium cordis, etc.) appears to be constituted by a rapid succession of inco-ordinated peristaltic contractions—a condition that can be brought about either (1) by the influence of certain depressing or paralysing agents upon the ventricular tissue, or (2) by the application of certain forms of stimulation to the ventricular tissue.

In the first class of cases the depressing influences alluded to probably throw out of gear the co-ordinating arrangements while they leave the muscular irritability intact—or it may be even augmented largely. Then the excitable (and probably highly rhythmic) muscle contracts, but its excitation instead of assuming the form of a normal beat becomes a peristaltic contraction wave along the complexly arranged and intercommunicating muscular bundles. And if the ventricular muscle is in an excitable state there would naturally occur a rapid series of such inco-

ordinated peristaltic contractions. For apart from the possibility of rapid spontaneous discharges of energy by the muscular fibres, there seems to be another probable cause of continued and rapid movement. The peristaltic contraction travelling along such a structure as that of the ventricular wall must reach adjacent muscle bundles at different points of time, and since these bundles are connected with one another by anastomosing branches, the contraction would naturally be propagated from one contracting fibre to another over which the contraction wave had already passed. Hence if the fibres are sufficiently excitable and ready to respond to contraction waves reaching them, there would evidently be a more or less rapid series of contraction in each muscular bundle, in consequence of the successive contraction waves reaching that bundle from different directions along its fibres of anastomosis with other bundles. Hence the movement would tend to go on until the excitability of the muscular tissue had been lowered, so that it failed to respond with a rapid series of contractions. Then there might be some isolated peristaltic contractions such as I have often seen after the cessation of the fibrillar movement.

In the second class of cases—when the fibrillar contraction is excited by stimulation (e.g., faradisation of the surface of the ventricles) there appears to be a condition of violent excitement set up in the muscular tissue. The excitation of the muscular fibres travels peristaltically, producing the characteristic movement; the inco-ordinated contraction of the various fibres may be most distinctly realised when the ventricles are held between the forefinger and thumb; there is a sort of wriggling sensation to be felt as the individual muscular bundles become hard and wiry while the contraction is passing over them in succession. The co-ordinating arrangements of the ventricles are powerless to regulate and guide the contractions; those co-ordinating arrangements are very possibly not paralysed nor rendered incapable of action, but they are temporarily superseded and rendered inoperative by the excessive state of excitement which pervades the muscular fibres—just as the cerebrospinal co-ordinating mechanism might be rendered impotent by strong local stimulation of the skeletal muscles. When the fibrillar movement having become less rapid has at length stopped—its duration depending on the excitability of the muscle—there ensues a pause.

Then there may be a recovery of the normal co-ordinated beat provided the fibrillar condition (and consequent blood stasis) has not lasted so long as to involve a paralysis or death of the co-ordinating mechanism.

When the last mentioned change has taken place, any beats that may occur are of the fibrillar character.

VIII. The phenomena resulting from faradic stimulation of the auricles differ in various respects from those seen in the ventricles.

The application of the current sets the auricles into a rapid flutter, the rapidity of which largely depends upon the excitability of the auricular

tissue and the strength of current employed. The movements are regular; they seem to consist of a series of contractions originating in the stimulated area and thence spreading over the rest of the tissue. The movement does not show any distinct sign of inco-ordination; it looks like a rapid series of contraction waves passing over the auricular walls. The difference between this appearance and that seen in the ventricles probably depends on the simpler structure and arrangements obtaining in the auricles.

The persistence of the movement after the discontinuance of the stimulating current varies according to the excitability of the auricular tissue and strength of current employed. In very excitable conditions the rapid movement lasts for a considerable time; in depressed states the movement ceases almost immediately after the stimulation has ended. The persistence after the use of a strong current is, *caeteris paribus,* usually very much greater than when a weak current has been employed to excite the fluttering action.

IX. The movements excited by faradisation in the auricles and ventricles differ very markedly in their relation to the inhibitory influence of the vagus nerve. The fibrillar movement in the ventricles appears to be entirely unaffected by vagus stimulation; the fluttering movement of the auricles can be checked or arrested by the influence of the vagus.

Sometimes, when the auricles are very excitable, the fluttering movement is entirely suspended during vagus stimulation only to reappear when the inhibitory influence has passed away. The vagus influence appears to act by weakening the individual contractions to the point of invisibility. At other times the contractions are markedly weakened without being rendered invisible. Often the movement is entirely arrested and does not recur; the normal action of the auricles goes on after the period of inhibition has passed.

The relation of the vagus nerve to the auricular muscle seems to be entirely different from the relation of that nerve to the ventricular muscle.
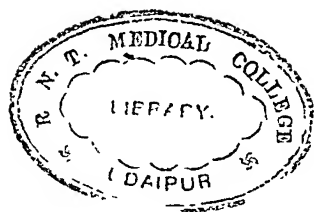
1888

GRAHAM STEELL

DESCRIPTION OF THE MURMUR OF HIGH PRESSURE
IN THE PULMONARY ARTERY, LATER TO BE
KNOWN AS THE GRAHAM STEELL MURMUR

GRAHAM STEELL

(1851-1942)

We regret our inability to include in this volume a biographic sketch of Dr. Graham Steell. Dr. Steell has granted us permission to reproduce his paper on "The Murmur of High-Pressure in the Pulmonary Artery," but he has requested that we do not attempt to delineate the leading incidents in his life and career. We are happy to record that Dr. Steell is still living. He was born on July 27, 1851.†

THE MURMUR OF HIGH-PRESSURE IN THE PULMONARY ARTERY*

By

GRAHAM STEELL, M. D.

Assistant Physician to the Manchester Royal Infirmary

THERE has long been question among physiologists of a safety-valve action of the tricuspid valves. Few clinicists will deny that, in disease, a similar occurrence takes place on the left side of the heart, the mitral valves becoming incompetent when the left ventricle is embarrassed under the effort which it is called upon to make. The muscle-element in the valve apparatus of the auriculo-ventricular orifices must be borne in mind in this relation, for it is by interference with muscle-action that incompetence of the valves is secured, and relief temporarily afforded in both cases. It is, therefore, the important part played by the heart-muscle in the establishment and maintenance of closure of both the tricuspid and mitral valves which renders possible the sudden production of their incompetence under special circumstances. In the case of the mitral valves, the causes which demand regurgitation in the way indicated have been, as a rule, long at work, and the accomplishment of regurgitation has been preceded by a series of changes. The valve-apparatus of the great arteries of the heart, unlike that of the auriculo-ventricular orifices, is independent of muscle-action, so that an analogous safety-valve action, in its case, appears to be out of the question. In health I believe it to be so, and, at the same time, I do not hesitate to express my disbelief in the rupture of a sound valve. In disease it is otherwise, and the clinical study of arterial high tension, aortic dilatation, and final incompetence of the valves, forces me to the admission that the arterial valves, like the auriculo-ventricular, do, under the strain

†[Dr. Steell died in 1942.]

of extreme tension long continued, permit of regurgitation through them. Thus there occurs an action analogous to a safety-valve one, although the name is less appropriate, since there is no threatened asystole to be obviated in their case, as there is in that of the mitral valves, inasmuch as the recoil or systole, of the elastic arteries is not a vital action. It is not my purpose here to discuss aortic regurgitation arising from dilatation, and to trace its origin directly and apart from induced disease of the valves, to high arterial tension. I wish to plead for the admission among the recognized auscultatory signs of disease of *a murmur due to pulmonary regurgitation, such regurgitation occurring independently of disease or deformity of the valves, and as the result of long-continued excess of blood pressure in the pulmonary artery.*

In cases of mitral obstruction there is occasionally heard over the pulmonary area (the sternal extremity of the third left costal cartilage), and below this region, for the distance of an inch or two along the left border of the sternum, and rarely over the lowest part of the bone itself, a soft blowing diastolic murmur immediately following, or, more exactly, running off from the accentuated second sound, while the usual indications of aortic regurgitation afforded by the pulse, etc., are absent. The maximum intensity of the murmur may be regarded as situated at the sternal end of the third and fourth intercostal spaces. When the second sound is reduplicated, the murmur proceeds from its latter part. That such a murmur as I have described does exist, there can, I think, be no doubt. Let me quote, with regard to it, the testimony of my revered master, Dr. G. W. Balfour, though he gives a very different explanation of the murmur from that which I advocate. Speaking of the rare occurrence of pulmonary incompetence from disease of the valves, he says:* "I mention it just now mainly for the purpose of warning you against being led into mistaking an auricular diastolic murmur for a pulmonary diastolic one. I have already pointed out that mitral stenosis is not infrequently associated with a diastolic murmur apart and distinct from its own peculiar presystolic murmur. Now and then this diastolic murmur of auricular origin has its position of maximum intensity at the sternal end of the fourth rib, a position in which it might readily be mistaken for a pulmonary diastolic murmur, and possibly has been so mistaken." In another place, he speaks of the position of maximum intensity being "frequently in the pulmonary area."† I must here remark, that the murmur, which I have described, is altogether different from the obstructive diastolic murmur of mitral stenosis, which is essentially an apex murmur, and, moreover, is wanting in the soft blowing quality of the pulmonary regurgitant murmur. The mitral murmur, too, runs off from the first part of a reduplicated second sound, the pulmonary from the last part.

*Clinical Lectures on Diseases of the Heart and Aorta, p. 218.
†Note, p. 119.

I am prepared for the objection that the murmur under consideration is only the murmur of a slight amount of aortic regurgitation, the usual evidence of which in the pulse is masked by the mitral lesion. How difficult it is to distinguish between the murmurs of aortic and pulmonary regurgitation respectively, by means of auscultation alone, will be admitted when it is remembered that the ordinary murmur of aortic regurgitation is probably conveyed to the surface directly through the right ventricle, and especially through that part of it which is called the conus arteriosus or infundibulum. With reference to this fact, the late Doctor Gibson's description is interesting. He says:* "The root of the aorta, including its orifice, valve, and sinuses, occupies the space between the pulmonic and tricuspid orifices. The root of the aorta and the aortic vestibule, which is the channel or chamber with rigid walls that leads to it from the cavity of the left ventricle, project forwards in front of that cavity and of its mitral orifice, so that the orifice of the aorta, covered by the posterior wall of the conus arteriosus, interposes itself, as has just been stated, between the pulmonic and tricuspid orifices. By this arrangement the aortic orifice advances more nearly to the front of the chest, the shallow conus arteriosus being in front of the orifice, and the deep cavity of the right ventricle being below it. Hence the murmur of aortic regurgitation, and an intensified aortic second sound, and coincident doubling of that sound, are heard loudly over and to the left of the middle third of the sternum in front of the arterial cone and the root of the aorta." When there is pulmonary regurgitation, again, the conus arteriosus will be the seat of the fluid veins which produce the diastolic murmur, and over it, accordingly, we may expect to find the maximum intensity of the murmur. The fact that in mitral stenosis the conus arteriosus is generally enlarged, and therefore extends to the left of the sternum to a greater degree than in the normal condition, does not seem to offer an entirely satisfactory explanation of the peculiar localization of this murmur, supposing it to be an aortic regurgitant murmur, almost wholly to the left of the sternum. I must admit, however, that in cases in which a diastolic murmur was audible over the lower half of the sternum, and the other evidence in favor of aortic regurgitation was strong, I have often been struck by the loudness of the murmur to the left of the sternum. Possibly enlargement of the right ventricle, with extension of the conus arteriosus to the left, was present in all these cases.

The murmur of high-pressure in the pulmonary artery is not peculiar to mitral stenosis, although it is most commonly met with, as a consequence of this lesion. Any long-continued obstruction in the pulmonary circulation may produce it. The pulmonary valves, like the aortic, do not readily become incompetent, apart from structural change. Probably no

*Reynold's System of Medicine, Vol. IV, p. 84.

amount of blood pressure in the pulmonary artery will render them so suddenly, as, at least, theoretically, the mitral valves may be rendered incompetent. Changes in the vessel, with widening of its channel, and, eventually, of its orifice, long precede the occurrence of incompetence of its valves. The pulmonary murmur of high-pressure is probably never persistent at first, and one of its most remarkable features is, as a rule, its variableness in intensity. On some days it will be distinctly heard, on others, it will be indistinct, or even inaudible; while extreme accentuation of the pulmonary second sound is always present, the closure of the pulmonary semilunar valves being generally perceptible to the hand placed over the pulmonary area, as a sharp thud. This non-persistence of the murmur, in the earlier stages, at any rate, is only what the study of dilatation of the aorta and the consequent regurgitation would lead us to expect. Indeed, so common is a soft, blowing murmur, after an accentuated aortic second sound, that extreme accentuation should make us listen, with special care, for a murmur, and even though it be absent on the first occasion the search should not be abandoned. My belief is, that when the aortic second sound is extremely accentuated, regurgitation, to some extent, will probably occur sooner or later. Its supervention in aneurism of the first part of the arch of the aorta is a familiar fact. Post mortem, enlargement of the left ventricle, in these cases, may be a better indication of regurgitation having occurred during life than the usual test of filling the cut aorta with water, a proceeding which cannot imitate the action of the forcible blood-currents in the living body. An accentuated second sound is no way incompatible with a certain amount of incompetence of the semilunar valves; on the contrary, an accentuated second sound, associated with a regurgitant murmur, is clinically common.

Writing in 1881,* after describing the regurgitant murmur of aortic dilation, I referred to the murmur which is the subject of this paper, as follows: "I am inclined to believe that a murmur of similar mechanism occurs on the right side of the heart, when there is much obstruction to the pulmonary circulation, with a dilated pulmonary artery." My subsequent experience has only served to confirm the opinion thus cautiously expressed more than seven years ago, though my faith has from time to time been shaken by a case presenting a murmur which I had at first imagined to be an example, but which, on further investigation, proved to be of aortic origin.

At a meeting of the Clinical Society of London,† on January 27th, 1888, Sir Dyce Duckworth related a case of "tricuspid and mitral stenosis in which physical signs of pulmonary arterial reflux were present," and stated his belief that the pulmonary arterial reflux was probably

*The Physical Signs of Cardiac Disease, Maclachlan and Steward, Edinburgh, 1881.
†British Medical Journal, February, 1888.

explicable by the dilated state of the vessel which was found after death. In this case the murmur was not persistent, in this respect corresponding to the description given above.

Only a few cases of incompetence of the pulmonary valves from structural change or deformity are on record. One of these was related by the late Dr. J. Warburton Begbie, who described the site of the murmur in the following words: "On more careful examination, the thrill was found to be almost entirely limited to the situation in which a loud systolic murmur was heard with the greatest degree of intensity. That was at the left border of the sternum, over the cartilage of the third rib. The systolic murmur thus distinguished was blowing in character and of unusual loudness; in the same situation it was followed by a diastolic murmur of much less intensity. The systolic murmur was readily distinguished over the whole upper part of the chest, but with much facility the seat of its greatest intensity was determined to be that already indicated. The diastolic murmur was limited, or almost limited to the same situation."[*]

Doctor Hayden[†] writes:—"From aortic diastolic murmur that of pulmonary inadequacy may be distinguished, not only by its seat of origin being to the left of the sternum, but likewise, as urged by Doctors Gordon and Begbie, by the absence of visible pulsations in the arteries."

Doctor Hope[‡] wrote:—"Diastolic murmur of the pulmonic valves . . . I created this murmur artificially in an ass poisoned with woorara, by making a perforation through one valve. We found the murmur soft, prolonged, and audible down the ventricle, exactly as in aortic regurgitation. In the human subject the pulmonic would probably be louder than the aortic diastolic murmur, because its seat is nearer the surface."

Doctor Bramwell[§] writes:—"Pulmonary incompetence is attended with a diastolic murmur, which has its point of differential maximum intensity in the pulmonary area, and its direction of propagation downwards to the right. The murmur, like the diastolic murmur of aortic regurgitation, would probably in many cases, be best heard at the lower end of the sternum."

The limitation of the murmur of pulmonary regurgitation to the left of the sternum does not seem to be in accordance with theoretical considerations, for we should expect the murmur to be conducted, as Doctor Bramwell says, "downwards and *to the right,*" over the right ventricle. Apparently this is not the case, as a rule, and when the murmur is conducted to the lowest part of the sternum its maximum intensity will always be found to the left of the bone.

[*]Beale's Archives of Medicine, Vol. II, page 11.
[†]Diseases of the Heart and Aorta, p. 1,005.
[‡]A Treatise on the Diseases of the Heart and Great Vessels, 3rd edition, p. 76.
[§]Diseases of the Heart and Thoracic Aorta.

With regards to prognosis, the murmur probably points to the vigour of the right ventricle being well maintained.

The discrimination of the murmur in question from the murmur of aortic regurgitation will always be a matter of peculiar difficulty, from the reasons already indicated; but it is surely of some importance that conditions so dissimilar as arterial regurgitation on the right and on the left sides respectively of the heart should be distinguished at the bed side.

In matters medical there should not even be the semblance of special pleading. Let me, therefore, in conclusion, distinguish between the facts observed, and the theory advocated to explain them. It is simply a matter of experience, that in a considerable proportion of typical cases of mitral stenosis there is audible a murmur with the characters and in the situation described above, while in the same cases the left ventricle is not enlarged* and the pulse does not possess the peculiarities which we know to be produced by aortic regurgitation. Shall we explain the fact by saying that the mitral stenosis prevents the enlargement of the left ventricle, which would otherwise result from aortic regurgitation, and at the same time interferes with the due development of Corrigan's pulse; and moreover that in these cases the leakage through the aortic valves is trifling in amount? We may do so plausibly enough. Or shall we agree with the late Doctor Fagge, whose every sentence deserves attention, in his statement with reference to a case of pulmonary regurgitation from disease of the valves, in which case the great rarity of the disease led to its rejection as a diagnosis.† "Indeed one can hardly expect in future to attain to greater accuracy; for (as we shall presently see) the pulse may fail to be characteristic of aortic regurgitation even when this disease exists; and the tendency of aortic diastolic murmurs to be transmitted downwards along the sternum, must always prevent a pulmonary regurgitant murmur from being identified by its being heard over the right ventricle," and confess that, under the circumstances, we cannot distinguish an aortic from a pulmonary regurgitant murmur? To those to whom neither the dogmatic assertion that all such murmurs as I have described, are, in spite of the absence of any confirmatory evidence, aortic in origin, nor the sceptical *non possumus* of Doctor Fagge, commends itself, I would urge a careful consideration of "the high-pressure murmur of the pulmonary artery" as a feasible explanation. I have stated my own opinion; from others I ask only that this murmur should find a place—however subordinate—among the physical signs of disease, which they recognise.

*We occasionally meet with an enlarged left ventricle in a case of apparently *pure* mitral stenosis. The cause of such enlargement was long a mystery to me, but I now believe that it is produced at an early period of the disease, while mitral regurgitation was the only or predominant effect of the valve changes.

†Reynold's System of Medicine, Vol. IV, p. 646.

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†Reynold's *System of Medicine*, Vol. IV, p. 646.

1888

ÉTIENNE-LOUIS ARTHUR FALLOT

DESCRIPTION OF THE "MALADIE BLEUE," LATER TO
BE KNOWN AS THE TETRALOGY OF FALLOT

ÉTIENNE-LOUIS ARTHUR FALLOT

(1850-1911)

ÉTIENNE-LOUIS ARTHUR FALLOT was born in Cette, France, on September 29, 1850. He was educated at the Lycée at Marseille, where he received a prize for high scholastic ability. He studied medicine at the École de Médecine at Marseille. After graduation he served as substitute professor of medicine at the University of Marseille from 1882 to 1886. In 1886 Fallot took charge of the course of pathologic anatomy. He continued in this capacity until 1888, in which year he was appointed professor of hygiene and legal medicine. He continued in this post until his death on April 30, 1911.

Because of Fallot's request that no eulogy be written after his death, little biographic information concerning him is available. Fallot's memory is perpetuated, however, by his studies on the congenital affections of the heart. He described with precision the most common anatomic type. He demonstrated in 1888 that the cardiac lesions of the "maladie bleue" (morbus caeruleus) may be summed up in a characteristic tetralogy which has since been called by his name. We are reproducing in translation Fallot's summary of this important work.

CONTRIBUTION TO THE PATHOLOGIC ANATOMY
OF MORBUS CAERULEUS (CARDIAC CYANOSIS)*

By

DR. A. FALLOT

. . . .

Here we conclude the presentation of the results of our investigations; not that we pretend to have covered the subject completely, or to have discussed all of its details, but because we have reached the limit that we set at the beginning. The following lines briefly summarize the conclusions which we believe may be drawn from our study.

I. Until now, clinicians have considered the precise diagnosis of anatomic lesions of morbus caeruleus of almost unsurmountable difficulty, as if it could be pronounced only as a vague and uncertain hypothesis. On the contrary, we see from our observations that cyanosis, especially in the adult, is the result of a small number of cardiac malformations well determined.

II. One of these cardiac malformations is much more frequent than the others, since we have found it in about 74 per cent of our cases; this is what the clinician should diagnose and in doing so, his chances of error are relatively slight.

III. This malformation consists of a true anatomopathologic type represented by the following tetralogy· (1) Stenosis of the pulmonary artery; (2) Interventricular communication; (3) Deviation of the origin of the aorta to the right; (4) Hypertrophy, almost always concentric, of the right ventricle.—Failure of obliteration of the foramen ovale may occasionally be added in a wholly accessory manner.

IV. One cannot, at present, attribute cyanosis to the persistence of the foramen ovale without putting himself in explicit opposition to the great majority of observed facts; communication of the two auricles, when it exists without any other concomitant cardiac lesion, does not produce cyanosis.

V. From the historical point of view one finds, with the writers of the last century and of the beginning of the present one, a fair number of observations on cyanosis; for the most part they offer the interesting

peculiarity that the existence of the various cardiac lesions previously mentioned is met with and fully described.

VI. Finally, from the point of view of pathogenesis, the theory which considers interventricular communication as a simple phenomenon, appertaining to a group of reversive anomalies, rests only on a superficial and inexact interpretation of facts; in persons with morbus caeruleus, the incompletely developed septum cannot by any means be considered as the analogy of the false septum of vertebrates to communicating ventricles. It seems to be much more logical and more in conformity with the laws of physiology to regard the whole series of cardiac changes enumerated as wholly the result of pulmonary stenosis. As to the cause of this, we believe that we must attribute it not to a simple arresting of development but rather to a pathologic process developing during intra-uterine life at the level of the pulmonary valves and of the region of the infundibulum which is contiguous to them.

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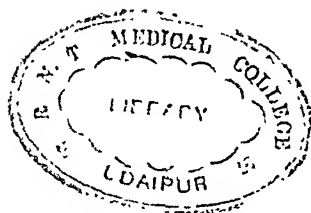
1893

WILHELM HIS, JR.

DESCRIPTION OF THE AURICULOVENTRICULAR BUNDLE (BUNDLE OF HIS)
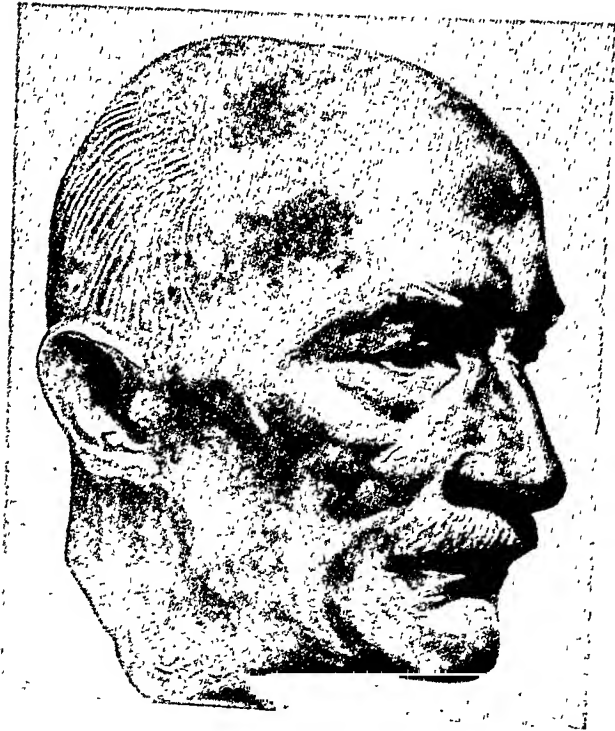
WILHELM HIS, JR.

WILHELM HIS, JR.

(1863-1934)

"The knowledge of the denervated beating heart and the significance of the nervous function is today as forty years before, a fascinating but incompletely solved problem."

—Wilhelm His, in 1933, at the age of seventy
(Klinische Wochenschrift).

ILHELM HIS, THE YOUNGER, was born in Basle, Switzerland, on December 29, 1863. His father at that time was professor of anatomy at the University of Basle. Wilhelm His, the elder, was called to the University of Leipzig in 1872. There he shared with Carl Ludwig the chair of anatomy vacated by Weber. Wilhelm Junior studied at the gymnasium in Leipzig. Leipzig was the center of culture in Southern Germany and the young student's outlook was broadened by his many contacts with music and the arts. Young His spent the last two years of his gymnasium at Basle. He studied at the Universities of Leipzig, Strasbourg, Bern, and Geneva. He was graduated from the University of Leipzig in 1888.

In 1889 he became an assistant in the medical clinic at Leipzig under Heinrich Curschmann. In this clinic Krehl was a first assistant and Ernst Romberg was a co-assistant. In 1893 a small volume entitled "Arbeiten aus dem medizinischen Klinik zu Leipzig" was published from the clinic. In addition to some fundamental studies by Krehl and Romberg there appeared in this work the first observations of His concerning the atrioventricular bundle of the heart and its function. We are presenting in translation His's summary, as recorded in this early paper. Of the experimental work which led him to his discovery, His wrote, in 1933:

"It was the current teaching of the time that the ganglia are the autonomic centers of the heart. Only Engelmann, in Utrecht, and Gaskell, in Cambridge, held the belief, based on their experiments, that the heart muscle itself is able to originate rhythmic stimuli. I was present one day when Krehl and Romberg discussed these subjects. I proposed to them to study from an embryological point of view the development of the heart to try to ascertain whether or not the heart is able to beat before it has nerves and ganglia. At that time I had finished an embryological paper under my father's direction and was, therefore, familiar with the technique necessary for such a study.

"I followed the development of the cardio-nervous system through several vertebrates and could prove that in all these animals the heart beats before it receives cerebral spinal nerves or ganglia. One point remained mysterious, namely, the conduction of the stimulus from one part of the heart to the other.

"Gaskell had shown that in the frog and turtle the conduction is made by way of the muscles. I tried to prove such a muscular connection in the adult mammal and in human beings by examining serial sections in the various embryonic stages. I finally found these muscular connections and described them in 1893. Few have read this paper."

In His's original paper of 1893 he also described the first instance of the occurrence of Adams-Stokes syndrome in Germany, and he was able to demonstrate the pathologic seat of the interrupted conduction in the bundle.

His, in 1891, became associate professor of internal medicine at the University of Leipzig. In 1901 he went to Dresden and in 1902 he succeeded Friedrich Müller as director of the medical clinic at Basle. At Basle, His became interested in physiologic chemistry and with his assistant, Bloch, he worked on the biochemic aspects of uric acid.

In 1907, His succeeded Ernst Victor von Leyden in Berlin. Here, he continued his studies on the heart and also gave much attention to the metabolism of gout and to diseases of the blood. In Berlin he was popular as a teacher. He also served for some time as editor of the "Zeitschrift für klinische Medizin," and later became director of the first medical clinic at the University of Berlin.

His had become a naturalized citizen of Saxony in 1895, so that at the outbreak of the World War he joined the German army as a voluntary soldier in spite of his being Swiss by birth. The German surgeon-general, Lieutenant-General Otto von Schjerning, created a special appointment for him, that of consulting internist on September 2, 1914, and he was assigned to the theater of the war in East Prussia. His observations on, and measures against, epidemics proved to be very valuable and von Schjerning sent him on missions to Turkey, Asia Minor, the Western theater of war, Russia and the Ukraine. During the war, he was the first to describe (February 23, 1916, at Warsaw) Volhynia fever (trench fever), which he named after a district in Russia in which he observed the disease. His's experiences in the war were set forth in a small book, "Die Front der Ärzte." This was published in 1931, and proved so fascinating that it had wide circulation outside of the medical profession. In 1933 the Association of Military Surgeons of the United States sponsored an English translation of the book by Dr. Gustavus M. Blech, of Chicago.

The outstanding characteristic of His's personality was a lofty standard of education and culture. He was a good violinist, a talented painter, and had a keen appreciation of art, literature, and history.

His suffered from emphysema and died on October 10, 1934.

THE FUNCTION OF THE EMBRYONIC HEART AND ITS SIGNIFICANCE IN THE INTERPRETATION OF THE HEART ACTION IN THE ADULT*

By

DR. WILHELM HIS, Jr.

. . . .

After extensive investigation I was able to find a muscle bundle which connects the auricular and ventricular septal walls, and which apparently had not been observed before, because it is only visible in its entire distribution when the septal walls are cut exactly in the horizontal direction. I was able to recognize the course of this bundle on such sections and on serial sections, and have proved its presence in an adult mouse, in a new-born dog, in two new-born infants and one adult, about 30 years of age. The bundle arises from the posterior wall of the ventricle near the auricular septum in the atrioventricular groove; it joins the upper edge of the ventricular septum and ramifies, coursing on the septum anteriorly until it branches near the aorta into a right and a left branch, the latter terminating in the base of the aortic cusp of the mitral valve.

I cannot state with certainty whether this bundle actually conducts the impulses from the auricle to the ventricle, as I did not perform any experiments dealing with the severing of the bundle. Its presence, in all events, is contrary to the opinion of those, who, in the absence of such a muscular connection between the auricle and ventricle, attempt to prove the necessary presence of a nerve conduction.
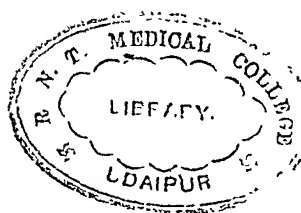
1896

FRANCIS H. WILLIAMS

FLUOROSCOPY OF THE HEART

FRANCIS HENRY WILLIAMS

(Courtesy American Journal of Roentgenology and Radium Therapy.)

FRANCIS HENRY WILLIAMS

(1852-1936)

*F*RANCIS HENRY WILLIAMS, a pioneer in the development of roentgen-ray and radium therapy, was born in Uxbridge, Massachusetts, the son of Henry Willard and Elizabeth Dew Williams. He attended the Massachusetts Institute of Technology and was graduated from that institution in 1873. The following year, he traveled around the world. During this trip he attended a meeting in Japan as a member of the United States Transit of Venus Expedition.

Following his journey, Williams began the study of medicine at Harvard University, and in 1877 he received the degree of Doctor of Medicine from that institution. He spent the next two years in Europe in postgraduate study.

In 1879 he established himself in Boston and began the practice of medicine, interesting himself particularly in diphtheria. In Boston he spent his long and useful career. In 1884, he was appointed instructor in materia medica in the Harvard Medical School, and he later became assistant professor of therapeutics at that school.

In 1891 he was married to Anna Dunn Philips, of Boston.

Williams first began his work on the roentgen rays in 1896 (only a year after their discovery) while he was visiting physician to the Boston City Hospital. Because the Boston City Hospital at that time did not possess the necessary facilities, his first patients were examined at the Rogers Laboratory of Physics of the Massachusetts Institute of Technology. Eventually, the trustees of the Boston City Hospital granted him quarters in the basement and there he worked until 1915. In 1913, with the establishment of the roentgen-ray department at Boston City Hospital, he was appointed senior physician.

Williams was quick to observe the diagnostic value of the roentgen rays. We are reprinting his important article, which was one of the first published accounts of fluoroscopy of the heart (1896). Therein, in addition to showing how pathologic conditions may be observed by means of the fluoroscope, he suggested correlating roentgen-ray studies with data obtained by other means of physical diagnosis.

By 1898, through the medium of the fluoroscopic screen, Williams was able to demonstrate the application of the roentgen rays to the diagnosis of thoracic aneurysm, pericardial effusion, cardiac hypertrophy, cardiac transposition, emphysema, pleurisy with effusion, pneumothorax, including hydropneumothorax, and pulmonary tuberculosis.

In 1899 Williams and Walter B. Cannon, with the aid of the fluoroscope, demonstrated important physiologic facts concerning the stomach and intestines. In two children, they were able to show the relationship of visceral position to suggestive posture, the excursion of the stomach during respiration, and the changes in its shape during digestion.

In 1901, Williams published his comprehensive work, "Roentgen Rays in Medicine and Surgery." This was a successful publishing venture and a second edition appeared in 1903.

With his friend, Dr. William Rollins, Williams perfected new instruments to aid the practical use of roentgen rays and radium. Chief among these was the invention

of the fluorometer in 1902. This instrument is used for measuring the quantity of roentgen rays given out by the roentgen-ray tube. It has also been used for the quantitative estimation of beta particles (electrons) and gamma rays given off by the radium (and radioactive) salts. Williams and Rollins also improved on the fluoroscopic screen and invented a mechanical stereoscopic fluoroscope.

Dr. Williams was the recipient of many honors. He was a fellow of the American Association for the Advancement of Science, and a fellow of the American Academy of Arts and Sciences. He served as president in 1917-1918 of the Association of American Physicians. He was an active member of the Massachusetts Medical Society, the American Medical Association and the Société de Radiologie médicale de France. He was a corresponding member of the K. K. Gesellschaft der Ärzte in Vienna, and an honorary member of the American Radium Society, the American Roentgen Ray Society, and the Radiological Society of North America.

It is significant, as Percy Brown has pointed out, in "American Martyrs to Science Through the Roentgen Rays," that Dr. Williams realized the dangers inherent in roentgen rays almost from the beginning of his career. Brown quotes Williams as saying:[1] "I thought that rays having such power of penetrating matter, as the x-rays had, must have some effect upon the system, and therefore I protected myself." So far as is known, Williams never was afflicted by the terrible ulcerative and carcinomatous processes that attacked other pioneer workers who did not venture to protect themselves against the roentgen rays.

Dr. Williams retired from active practice in 1930, at the age of seventy-eight. He continued to contribute to the literature of his field until his death in 1936. At the age of eighty-three he published a small volume entitled: "Radium Treatment of Skin Diseases, New Growths, Diseases of the Eye and Tonsils."

[1]Brown, Percy: *American Martyrs to Science Through the Roentgen Rays*, ed. 1, Springfield, 1936, Charles C. Thomas, p. 17.

A METHOD FOR MORE FULLY DETERMINING THE OUTLINE OF THE HEART BY MEANS OF THE FLUORESCOPE TOGETHER WITH OTHER USES OF THIS INSTRUMENT IN MEDICINE*

By

FRANCIS H. WILLIAMS, M.D.

A SHORT account of some of my work on the applications of x-rays in medicine was read at the meeting of the Association of American Physicians held in April last and has been published in their "Transactions." I now wish to speak further of some of the uses of the fluorescope in medicine, leaving a fuller discussion of them and of my observations relating to physiology and diagnosis, to a later time, when I shall hope also to describe the methods of examination that I have employed.

The picture which presents itself to the eye when the body is examined by the fluorescope is full of interest. The trunk appears lighter above than below the diaphragm and the rise and fall of the muscle are distinctly seen; the chest is divided vertically by an ill-defined dark band which includes the backbone; and each side is crossed by the dark outline of the ribs, the spaces between which, are the brightest portion of the picture. One also sees the pulsating heart, especially the ventricles, and under favorable conditions the right auricle and left auricle, but it is difficult to separate the latter from the pulmonary artery; a small portion of one side of the arch of the aorta may be seen in the first intercostal space to the left of the sternum. The organs of the abdomen are much less readily observed, but the presence of a piece of lead or of substances impermeable to the Röntgen rays may be detected in them. The neck and face may be reached with the fluorescope; and in the arms and legs the bones and certain foreign substances may be seen. The head is the least promising field.

In examining the heart by means of percussion, we can usually determine its left border, but we cannot find its lower border. Now let us see what can be done in this direction by means of the x-rays. The constant motion of the heart and diaphragm interferes with the use of radiography but renders fluoroscopy all the more valuable. The lungs and the organs adjacent to them are the parts of the body which best lend themselves to fluoroscopic examination, because of the great difference in

*Boston M. & S. J. 135: 335-337, 1896.

density between the former and the latter, or, in other words, of permeability to the x-rays. The lungs being less dense than the neighboring organs allow the x-rays to pass through them more readily, and thus appear light against a darker back-ground formed by the heart and parts of the liver and spleen, which, owing to their density, are less permeable by the rays and thus appear dark when seen through the fluoroscope, that is, there is contrast.

The heart lies in such a position, however, that ordinarily but a certain portion of its outline may be seen with a fluoroscope, a horizontal plane may be imagined through the body, when in a standing position, that would pass through the heart, liver and spleen, as these latter organs overlap the heart to some extent; but it is readily possible to isolate the heart, as it were, by the contraction of the diaphragm; the organs below the heart being then depressed, the overlapping is avoided and the heart being more closely surrounded by transparent lung tissue the whole of the apex, and part of its lower border come into view and may be drawn on the skin. A suitable position of the Crookes tube of course facilitates this end somewhat. It is desirable to see as much as possible of the heart at one time in order to best estimate its condition—then if necessary we may study one or another portion separately—and by means of this fluoroscopic examination we can follow a larger portion of its outline and gain more information as to its size, position and action than has hitherto been within our reach. I may add here that I have made an instrument that enables me to listen to the heart-sounds while watching the pulsating organ.

The character of the revelations which are made to us by a fluoroscopic examination of the heart may be most briefly suggested by Figs. 1, 2, 3 and 4, taken from photographs of lines traced on the skin, which follow the outlines of the organs as seen through the fluoroscope. The patients from whom these photographs were taken were lying on a canvas stretcher and the Crookes tube was placed under and about a foot below the trunk. These illustrations have been selected from a number of photographs I have thus far made.

CASE I. Fifty-seven years old. The Crookes tube was placed eighteen inches away from and under the point indicated by the black dot in Fig. 1. The heavy crossed lines indicate the sixth rib; the full lines, what was seen in the fluoroscope; the broken line, the border of the heart obtained by percussion, which on the left side coincides with the line as seen in the fluoroscope. The lower border of the heart cannot be got by percussion, and is seen in the fluoroscope only during deep inspiration. No apex beat was felt. The full parallel lines on either side of the body mark the diaphragm in ordinary expiration and deep inspiration respectively.

CASE II. Twenty-seven years old. The full curved line on the left (see Fig. 2), as far as the dot which marks the apex beat, and the broken line

inside, indicate both what was seen in the fluorescope, and what was obtained by percussion—the full line during ordinary expiration, the broken line during deep inspiration. The continuation of the full curved line (beyond the broken line) that runs towards the sternum marks the lower

Fig 1.

Fig. 2.

border of the heart, which as above stated, is obtained by the fluorescope, and this only during deep inspiration. The two parallel lines on the left mark the diaphragm—the upper in ordinary expiration, the lower in deep inspiration. The difference between these is greater than in Figs. 1 and 3.

CASE III. A boy, eleven years old. The lines in Fig. 3 indicate what was seen in the fluorescope. The full curved line on the left marks the border of the heart as seen during ordinary expiration; the broken line inside, during deep inspiration; the two parallel lines on either side, the diaphragm in ordinary expiration and deep inspiration; the black dot, the point where the apex beat was felt.

Fig. 3.

Fig. 4.

CASE IV. Fifty-eight years old. Enlarged heart; examination with the fluorescope: the left and part of the lower border of heart are seen (Fig. 4) and the diaphragm lines already alluded to in the other cases. The difference between these last mentioned lines is less than usual.

of water. Thus it is easy to understand why fluid in the pleural cavity dulls or obliterates the outline of the adjacent organs, when the lungs become dense by disease they may obliterate not only the outlines of the ribs, but also those of the liver, spleen, and heart, as I have already indicated. When there is not marked contrast between the intercostal spaces and the ribs, or when the outlines of the clavicle and of adjacent organs are not defined, it should always arouse suspicion of something abnormal in the lungs or pleura.

The application of x-rays to surgery has hitherto formed the prominent side of their usefulness; I have pursued my investigations believing that it would be possible to demonstrate their usefulness in medicine, as distinguished from surgery, and am now confident that the advances these x-rays render possible in medical diagnosis are great, and that they will prove a more valuable instrument in the hands of the physician than of the surgeon. I have found them especially useful in diseases of the heart and lungs. We may now look where we have previously only been able to listen, and sometimes to hear but imperfectly. The advance consists not alone in what we can now see that we could not see before, but also in that we can by hand and ear, and eye together strengthen and confirm these separate observations beyond their respective limitations; singly they are beams, together an arch which justifies a heavier weight of inference.

Most of the work here described has been done in the x-ray room at the Boston City Hospital; the cordial interest of the Trustees and of my colleagues on duty has facilitated greatly the carrying out of these observations, and I shall always be indebted to Prof. Charles R. Cross of the Massachusetts Institute of Technology and to the assistants in his laboratory for the opportunity of studying the physics relating to the x-rays.
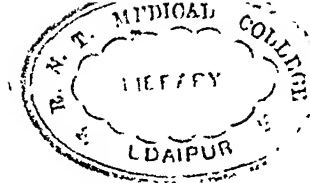
1897

SIR WILLIAM H. BROADBENT

ADHERENT PERICARDIUM

SIR WILLIAM HENRY BROADBENT

(Courtesy Charles C Thomas.)

SIR WILLIAM HENRY BROADBENT

(1835-1907)

"I trust in God from day to day, seeking and asking for nothing beyond the happy mean, neither poverty nor riches, but grace to serve Him and do the work He has given me to do."

—Sir William Henry Broadbent,
quoted in *The Practitioner.*

WILLIAM HENRY BROADBENT was born at Longwood, a small village in Yorkshire, England, on January 23, 1835. His father was a manufacturer of woolens and desired that his son should succeed him in business. Thus, after young Broadbent received some academic training at Huddersfield College, he left school at the age of fifteen to learn the fundamentals of his father's business. After a trial of two years, not finding the work to his liking, the future baronet embarked on a career in medicine, his choice of profession.

At the age of seventeen, Broadbent became an apprentice to a surgeon in Manchester. The apprenticeship was for a five-year period and the fee was to include the expenses of a medical course at Owens College. Broadbent was a brilliant student and in spite of his meager resources and the need for his daily visits to patients, dispensing medicines, and attending lectures at the Royal School of Medicine he won medals in botany, materia medica, anatomy, physiology, chemistry, midwifery, surgery, and operative surgery.

In 1856 Broadbent went to London for the first examination for the degree of Bachelor of Medicine at the University of London. There, too, he showed his intellectual superiority by winning gold medals in anatomy, physiology, and chemistry. In 1857 he passed the final examination of the Conjoint Board of the Royal Colleges of Physicians and Surgeons in London, and in 1858 he returned for his final examination for the degree of Bachelor of Medicine at the University of London. There he received the gold medal in obstetrics and came away with first-class honors in medicine.

As soon as he was qualified, he accepted the post of obstetric officer at St. Mary's Hospital in London. From this time until 1896 he was actively engaged in duties at St. Mary's. On the termination of his appointment as obstetric officer, Broadbent in 1859 became resident medical officer. The following year he was appointed pathologist to the hospital and also was chosen to lecture on physiology and zoology in the hospital's medical school. At a later date he became curator of the museum. In 1860, also, he received the degree of Doctor of Medicine from the University of London, and in the following year became a member of the Royal College of Physicians. In 1864, on the retirement of Dr. Chambers, Broadbent was appointed assistant physician.

At St. Mary's, Broadbent worked under Sibson, who at that time was a leading specialist on thoracic diseases. Sibson loved his specialty to such an extent that he was known to spend as long as half an hour listening to one murmur. Sibson was the source of Broadbent's inspiration in his subsequent work on diseases of the heart.

In 1871 Broadbent was promoted to be physician in charge of the in-patients at St. Mary's. He was also appointed lecturer in medicine, a position which he held for seventeen years. Broadbent was connected with many other hospitals in addition to St. Mary's. He served as physician to the London Fever Hospital from 1860 to 1879, and afterward acted as consultant. He was also on the medical staffs at Western General Dispensary and the New Hospital for Women.

Broadbent's output of original papers was large. These papers were later collected by his son, Dr. Walter Broadbent, and published in book form.[1] Although Broadbent was naturally interested in the whole field of internal medicine, he spent considerable time on the study of the nervous system and also the cardiovascular system.

An article that contributed to his early pre-eminence in the field of neurology was his paper on sensory-motor ganglia,[2] in which Broadbent explained the immunity to paralysis of bilaterally associated muscles in hemiplegia. The subject of aphasia was also of interest to Broadbent and he contributed several interesting articles on the condition. Broadbent also wrote on chorea, syphilitic affections of the nervous system, ingravescent apoplexy and alcoholic spinal paralysis.

Broadbent's great interest in cardiology is reflected in his lectures on "Prognosis in Valvular Disease of the Heart." He delivered these before the Harveian Society in 1884. In 1887 he delivered the Croonian lectures on the pulse before the Royal College of Physicians in London. These he elaborated upon in his book, "The Pulse," published in 1890 by Cassell and Company, London. This classic book shows what was accomplished with the use of the finger before the days of clinical employment of the sphygmometer.

In 1891, when Broadbent was invited to give the Lumleian lectures, he chose for his subject, "Prognosis in Structural Diseases of the Heart." These lectures as well as his Harveian lectures were the basis for his book on heart disease published in collaboration with his elder son in 1897. Included therein was Broadbent's first description of the signs of adherent pericarditis. This we are reprinting. According to his own account (published in 1898) Broadbent established this famous cardiac sign as early as 1868;[3] but it was not until 1895 when his son, Walter, made note of it in the "Lancet"[4] that it received its first publicity.

In 1892 Broadbent was appointed physician in ordinary to the Prince of Wales, who was later to become King Edward VII. The following year Queen Victoria conferred a baronetcy upon him on the occasion of the marriage of the Prince of Wales, whom Broadbent had attended two years previously for an attack of typhoid fever. Sir William was later named physician in ordinary to King Edward VII, and in 1901 he was created a Knight Commander of the Victorian Order.

Sir William was the recipient of many honorary academic degrees in recognition of his achievements. He was awarded the degree of Doctor of Laws by Edinburgh University in 1898, and by St. Andrew's University in 1899. In 1904 the University of Leeds made him an honorary Doctor of Science, and the University of Toronto granted him the degree of Doctor of Laws on the occasion of the meeting of the British Medical Association in the city of Toronto, in September, 1906.

Broadbent was one of the organizers and was also the first president of the Entente Cordiale Médicale (1904) and at the banquet given in Paris in honor of

[1]Broadbent, Walter: *Selections from the writings, medical and neurological, of Sir William Broadbent*, London, 1908, Frowde, Hodder & Stoughton, 444 pp.
[2]Broadbent, W. H.: An attempt to remove the difficulties attending the application of Dr. Carpenter's theory of the function of the sensori-motor ganglia to the common form of hemiplegia, Brit. & For. M. Rev. 37: 468-481, 1866.
[3]Broadbent, Sir W.: Adherent pericardium, Tr. M. Soc. London 21: 109-122, 1898.
[4]Broadbent, Walter: An unpublished physical sign, Lancet 2: 200-201, 1895.

English physicians he was invested with the Grand Cross and insignia of a Commander of the Legion of Honor of the Republic of France.

Apparently Sir William enjoyed good health most of his life. In 1906, however, he suffered a severe attack of pneumonia. This was followed by empyema, which was successfully relieved by operation, but from which he did not fully recover. He died on July 10, 1907.

Sir William Broadbent in 1863 had married Eliza Harpin, by whom he had two sons and three daughters. His elder son, Dr. John Francis Broadbent, succeeded him in the baronetcy. The younger son was Dr. Walter Broadbent, to whom we have also alluded.
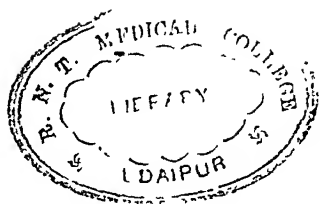
CHAPTER XVII

DISEASES OF THE PERICARDIUM*

ADHERENT PERICARDIUM

By the term "adherent pericardium" is implied the existence of adhesions between the visceral and parietal layers of the pericardium, the result of pericarditis. They may be limited to fibrous bands stretching across the pericardial cavity, or they may be universal, in which case the pericardium and heart are so intimately connected that the pericardial cavity is entirely obliterated. Adhesions may also exist between the chest-wall or pleura and the pericardium, as a result of so-called mediastino-pericarditis. The adhesions if of old standing are tough and fibrous, so that the pericardium cannot be stripped from the heart without tearing the heart-substance. There is also commonly some fibroid change in the heart-wall due to substitution of fibrous tissue for muscle fibres damaged by previous inflammation. In the case of recent adhesions or lymph undergoing organization into fibrous tissue, the two layers of pericardium on being separated will present a honeycomb or bread-and-butter-like appearance, owing to the layer of thick, sticky lymph which coats the surface.

Physical Signs

The physical signs differ according as the adhesions exist only between the two layers of the pericardium, or between the pericardium and chest-wall, or adjoining pleura as well. In the latter case they are more numerous and distinctive. Among them are the following:

Fixation of the apex beat, so that it does not alter its position in deep inspiration and expiration or in change of posture of the body.

Systolic depression of one or more intercostal spaces to the left of the sternum, or of the lower end of the sternum and the adjoining costal cartilages, which may be caused by the heart dragging on them at each systole, through the agency of the pericardial adhesions. The systolic recession of spaces alone is, however, not a trustworthy indication, as it may be due to atmospheric pressure, more especially when the heart is much hypertrophied. When the costal cartilages or lower end of the sternum are dragged in there can be little doubt as to the diagnosis, as this could not be effected by atmospheric pressure.

*Broadbent, Sir William H., and Broadbent, John F. H : *Heart disease with special reference to prognosis and treatment,* New York, 1897, William Wood & Co.

Systolic recession of the site of the apex beat is an important sign when a definite apex beat can be felt; when there is no palpable apex beat, systolic pitting over its site may be due to atmospheric pressure.

A diastolic shock may sometimes be felt on palpation with the flat of the hand over areas on the chest-wall where systolic recession is present. It is due to the elastic recoil of the chest-wall at the commencement of diastole as soon as the pulling force exerted during the systole ceases.

Systolic retraction of the lower portions of the posterior or lateral walls of the thorax may indicate the presence of a universally adherent pericardium. Such retraction may, however, be seen though the pericardium is not adherent to the heart, but only to a larger extent than normal to the central tendon of the diaphragm and the muscular substance on either side, and to the chest-wall as well. In such cases the heart is usually greatly enlarged and hypertrophied from old valvular disease. The explanation seems to be that the portion of the diaphragm to which the pericardium is adherent is dragged upwards at each systole of the heart, so that the points of attachment of the digitations of the diaphragm to the lower ribs and costal cartilages are dragged inwards and retracted.

The descent of the diaphragm in inspiration may be interfered with by pericardial adhesions between the heart and diaphragm, more especially if the pericardium is adherent to the chest-wall in front as well. This will be shown by impaired movement in respiration of the upper part of the abdominal wall in the epigastrium and left subcostal region.

The area of cardiac dulness will be increased, and will remain unchanged in inspiration and expiration, where there are extensive adhesions between the pericardium and chest-wall, as the lung, which normally overlaps part of the heart, will have been pushed aside, or perhaps have become involved in the adhesions, and be collapsed.

Enlargement of Heart.—It is common with adherent pericardium to find the heart, more especially the right ventricle, considerably enlarged, in the absence of valvular disease or other obvious cause to account for it.

It seems probable that such enlargement may be indirectly due to pericardial adhesions as follows: The heart becomes dilated during an attack of pericarditis, and, before it recovers its tone or can contract down again to its normal size, the pericardium becomes adherent and fixes it in this condition of dilatation, the right ventricle suffering more than the left, owing to its thinner walls, as well as for other reasons.

Hypertrophy and dilatation of the heart, more especially of the right ventricle, may therefore, in the absence of other obvious causes, such as valvular disease, high arterial tension, etc., to explain it, be a physical sign of considerable importance.

Diastolic collapse of cervical veins was held by Friedreich to be of great diagnostic value when accompanied by systolic retraction of spaces; but I have never found it to be of service.

The above remarks apply to the question of diagnosis in cases where, with or without valvular disease, there is no history of pericarditis, and the adhesions are of old standing.

In cases of pericarditis, which can be kept under observation after the attack, there will be less difficulty in arriving at a diagnosis, and the indications which would lead one to suspect that the pericardium was becoming adherent are as follows:—

1. Prolongation of the attack of pericarditis evidenced by a harsh friction rub over the praecordial area, which may persist for some weeks. When at the margins of the area of cardiac dulness a pleuro-pericardial friction is also heard, it will indicate that adhesions are probably taking place between the pericardium and adjoining pleura or chest-wall as well.

2. Permanent enlargement of the area of cardiac dulness to a marked extent after the subsidence of the pericarditis.

3. The occurrence of symptoms of right ventricle failure after a period of temporary improvement, there being no apparent exciting cause for the breakdown of the right ventricle. Damage to the cardiac muscle by fresh myocarditis may, however, be responsible, and should be first excluded.

Prognosis

When the heart remains normal in size, and there are no adhesions between the pericardium and chest-wall, the universal adherence of the pericardium to the heart may not in an adult tend to materially shorten life. When the heart is enlarged, or when the pericardium is also adherent to the chest-wall, the prognosis is more serious. When adherent pericardium exists as a complication of valvular disease, it is still more likely to prove fatal eventually, by so hampering the right ventricle as to prevent its recovery when once compensation has broken down. The detection of adherent pericardium has also an important bearing on prognosis, inasmuch as it affords presumptive evidence of fibroid change in the heart-wall, and therefore renders the outlook even more unfavourable.

Treatment

The discovery of adherent pericardium, when present, is important from the point of view of treatment, not because anything can be done to remedy or remove the pericardial adhesions, once they are formed, but because, when it is present, it will be necessary to impose additional restrictions on the patient, so that no undue risks may be run of upsetting the compensatory balance, which would only be restored with great difficulty.

1903

WILLEM EINTHOVEN

THE ELECTROCARDIOGRAPH
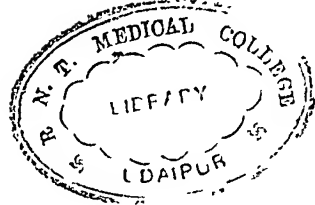
WILLEM EINTHOVEN

(Courtesy Heart.)

WILLEM EINTHOVEN

(1860-1927)

"Honours, however, were to him a smaller recompense than was the knowledge of the benefits which his long and arduous work had conferred upon his fellow men."

—Sir Thomas Lewis, on
Willem Einthoven *(Heart)*.

ONE OF the greatest advancements in modern cardiology was the conception of a means to study the electrical changes which accompany each heart beat. The development of electrocardiography and with it modern cardiology owes to Einthoven a great debt.

Willem Einthoven was born in May, 1860, in the Dutch East Indies (Semarang, Java), but spent most of his life in Holland. There he studied at the University of Utrecht from 1879 until 1885, in which year he received his Doctor's degree at the age of twenty-five.

Even as a medical student he had showed a remarkable interest in the physical sciences, but not until 1886, when his paper, ''On the Law of Specific Nerve Energies,'' was published, did his reputation in this field become established.

In 1886 Einthoven also published his inaugural thesis, ''On the Influence of Color Differences in the Production of Stereoscopic Effects.'' Einthoven's basic interest in the physical sciences is well reflected in this paper. In 1886, moreover, he was called to the chair of physiology and histology at the University of Leyden, in which post he was active for forty years until his death.

Einthoven contributed much to the understanding of the physiology of the bronchial musculature and to the knowledge of the physiology of the eye, but his greatest gift to medicine was his discovery of the string galvanometer. This pioneer contribution elucidated the physical principles of the electrocardiogram and enabled cardiologists to study, accurately, the biologic and physiologic activities of the heart.

At the end of the Eighteenth Century, Luigi Galvani (1737-1798), professor of anatomy at the University of Bologna, became interested in animal electricity. By accident one day he recorded a remarkable observation. A dissected frog had been placed on a laboratory table near an electric machine. Galvani's assistant had touched the nerves of the frog's leg with a knife, and the leg contracted vigorously. Galvani, intensely curious, investigated the phenomenon, finally discovering that the leg would contract in such a manner only when the electric machine was sparking. Leaman quoted Galvani of writing that ''strong contraction took place in every muscle of the limb and at the very moment when the sparks appeared.''

In many different experiments, Galvani later studied contractions of the muscles by using different methods of electrical stimulation, including lightning.

In 1842 Carlo Matteucci (1811-1868), another Italian, advanced the knowledge of electrical conduction. In an experiment he placed the cut, central end of the sciatic nerve of one leg of a frog on the muscles of the opposite leg. When he stimulated the

sciatic nerve on the intact side, both muscles contracted, although only the normal side had been stimulated. This observation was important in advancing knowledge of the influence of the electric current on nerves and muscles.

In 1856 Albert von Kölliker and Müller discovered that an exposed frog's heart produced an electric current which accompanied each beat. In 1878 John B. Sanderson and F. J. M. Page in England were finally able to record by means of the capillary electrometer, for the first time, the current produced by the action of the heart.

The next advance in the development of electrocardiography was contributed by Augustus D. Waller, who published a paper in 1887 entitled "A Demonstration on Man of the Electromotive Changes Accompanying the Heart's Beat."[1] This paper we reprint on pp. 656-661. Waller showed that the currents of the heart could be studied without opening the thorax of laboratory animals, that these studies could be made by connecting the surface of the body of such animals with electrodes to which the capillary electrometer was attached. These connections afterward were known as leads. Waller also demonstrated that the current of the heart in human beings could be studied in the same way.
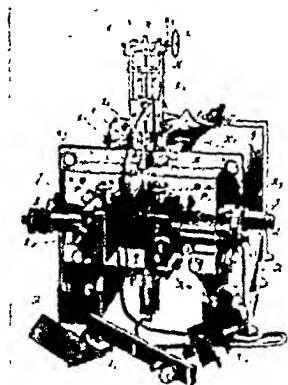
Einthoven's string galvanometer

(Courtesy Ciba Symposia.)

Much difficulty, however, was encountered with the capillary electrometer. This instrument consisted of a column of mercury contained in a vertical glass tube, one end of which was dipped into sulfuric acid. The mercury in this instrument would be disturbed by the electrical charge passing through it and the up and down movements of the mercury were photographed on a moving sensitized plate. Since the mercury, because of its inertia, produced curves which were not exact, the resulting recordings were not accurate measurements of the electrical changes accompanying the heart beats.

Einthoven, in 1903, employed the galvanometer, the invention of Johannes S. C. Schweigger (1779-1857), of the University of Halle, to measure the electric current

[1] J. Physiol. 8: 229-234, 1887.

produced by the action of the heart. By the use of his string galvanometer, Einthoven perfected Schweigger's invention, which meant that he introduced a practical method for electrocardiography.

Einthoven described the physical principles of the electrocardiogram and made pioneer contributions to the physiologic uses in which it may be utilized.

Soon Sir Thomas Lewis began to use the instrument in England and to correlate clinical data with observations arising from use of the new instrument, and the electrocardiogram thus became an indispensable aid of modern cardiologists.

In 1924, Willem Einthoven was awarded the Nobel Prize in medicine for his contributions which so greatly advanced the scientific study of modern cardiology and which are as important, perhaps, to the cardiologist as the use of digitalis is to the cardiac patient.

THE GALVANOMETRIC REGISTRATION OF THE HUMAN ELECTROCARDIOGRAM, LIKEWISE A REVIEW OF THE USE OF THE CAPILLARY-ELECTROMETER IN PHYSIOLOGY[1]*

By

W. EINTHOVEN

Physiologic Laboratory in Leyden

UP TO the present time, the human electrocardiogram discovered by Aug. D. Waller[2] could be recorded only by means of the capillary-electrometer. Simple inspection of the curve inscribed by means of this instrument results in an entirely fallacious representation of the variations of potential, which, as a matter of fact, actually existed. If one desires accurate values of the latter, the form of the registered curve must be corrected for the size of the capillary tube used, the degree of magnification, and the speed of the photosensitive plate. By this method one arrives at the construction of a new curve, the outline of which actually represents the variations of potential.

In explanation of this fact, the following example will be offered.[3]

Fig. 1 represents the registered curve of Mr. v. d. W. by leads from the right and left hand, while Fig. 2 represents the corrected curve.

The differences are obvious. One immediately likens the deflections C and D in the registered curve to the corresponding deflections R and T in the constructed curve. Only the latter portrays an accurate comparison of the height of the deflections.

What holds true for the electrocardiogram, also, in general, holds true for any other curve obtained by the capillary-electrometer, if one wishes to reproduce different changes in potential as occurring rather rapidly. One is obliged in both instances, regardless of the method[4] employed, to

*Einthoven, W.: Die galvanometrische Registrirung des menschlichen Elektrokardiogramm, zugleich eine Beurtheilung der Anwendung des Capillar-Elektrometers in der Physiologie, Pflüger's Arch. f. d. ges. Physiol. 99: 472-480, 1903. Translation by F. A. W.
[1]An investigation sponsored by the International Committee for the Unification of Physiologic Methods.
[2]Phil. Trans. Roy. Soc., London, vol. 180, p. 169-194, 1889.
[3]Pflüger's Arch., Bd. 605, 101, 1895.
[4]As shown on the preceding pages, the practical methods of electrocardiography result in curves which may easily be analyzed in a graduated rectangular coordinating system; see W. Einthoven, Lippmann's Capillar-Elektrometer zur Messung schnell wechselnder Potentialunterschiede. Pflüger's Archiv, Bd. 56, S. 528, 1894—Id., Ueber den Einfluss des Leitungswiderstandes auf die Geschwindigkeit der Quecksilberbewegung in Lippmann's Capillar-Elektrometer. Pflüger's Archiv Bd. 60, S. 91, 1895—Id., Ueber die Form des menschlichen Elektrokardiogramms. Pflüger's Archiv Bd. 91, S. 101.—Even earlier, other capillary-electrometer curves were analyzed showing circular zero lines, that were taken on a sensitive plate fastened to a pendulum. See G. J. Burch, On a method of determining the value of rapid variations of a difference of potential by means of the capillary electrometer. Proceed. of the Royal Soc. of London vol. 48, p. 89, 1890—Id. On the time relations of the excursions of the capillary electrometer. Philos. Trans. of the Royal Soc. of London vol. 183 A, p. 81, 1892—J. Burdon-Sanderson, The electrical response to stimulation of muscle. Part II. The Journal of Physiol. vol. 23, p. 325, 1898—Eine Vereinfachung der Ausmessung in rechtwinkligen Coordinatensystem findet man bei S. Garten, Ueber ein einfaches Verfahren zur Ausmessung der Capillarelektrometer-Curven. Pflüger's Archiv Bd. 89, S. 613, 1902. In the last paper a review of the other collections of measurements is given.

construct a new curve from the one originally inscribed, whereby accurate measurements can be determined, which are chiefly concerned with different calculations.

I have sought a method in which, as far as possible, the construction of a new curve could be avoided, and finally, to offer an instrument which primarily would satisfy the requirements of inscribing the electrocardiogram of human beings in approximately, at least, its correct proportions.

This instrument—the string galvanometer—is essentially composed of a thin silver-coated quartz filament, which is stretched like a string, in a strong magnetic field. When an electric current is conducted through this quartz filament, the filament reveals a movement which can be observed and photographed by means of considerable magnification, this movement is similar to the movements of the mercury contained in the capillary-electrometer. It is possible to regulate the sensitivity of the galvanometer very accurately within broad limits by tightening or loosening the string.
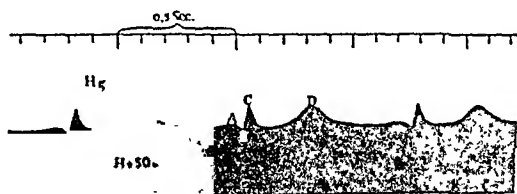
Fig. 1.
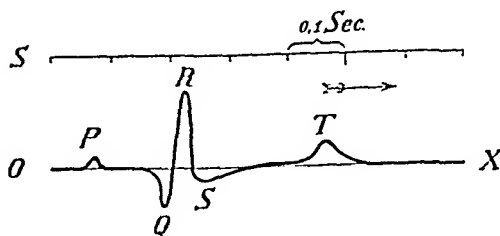
Fig. 2.

The theory, as well as the practical details, of this new instrument may be omitted at this time.[5] It is not only of interest to discuss the similarity of the capillary-electrometer to this new instrument but also certain characteristics by means of which the string galvanometer distinguishes itself.

It obviously is necessary to appreciate the fact that the movement of the string galvanometer measures electrical current, while the movement of the capillary-electrometer measures electrical potential. Here I will state that each time that variations in potential or strength of current

[5]See W. Einthoven, Ein neues Galvanometer, Drude's Annalen der Physik. Also in Onderzoekkingen Physiol. Laborat. Leyden, 2 Reihe, Bd. 5.

occur, the mercury meniscus, as well as the filament, is in motion. And during the phase of activity the capillary-electrometer must become alternately charged and discharged, while the string in the magnetic field develops an opposite electromotive force.

In addition, under conditions of constant and great resistance, together with negligible induction, the strength of the current must at any moment be proportional to the effective electromotive force, such as is usually the case in electrophysiology. Therefore, the differentiating principles of the electrometer and the galvanometer do not prevent the comparison of the two instruments.

The string galvanometer, as compared with the capillary-electrometer, offers various advantages.

1. In the first place, there are many instances, but particularly in the registration of the electrocardiogram of human beings, in which the deflections occur with more perfect aperiodicity, greater rapidity and with greater amplitude than those occurring with the capillary-electrometer. The greater sensitivity of the string galvanometer must be ascribed partly to the fact that the finer quartz filament, in spite of its greater length, is nevertheless considerably lighter than the mercury column of the capillary-electrometer. A quartz filament can be 10 times thinner than a mercury column; thus it is possible to have one with a cross section 100 times smaller, because the specific gravity of molten quartz is approximately 5 to 6 times less than that of mercury. From this, one must calculate the active force of the moving quartz filament and take into consideration the fact that only the mid-portion, when observed microscopically, shows the greatest displacement. All other portions of the filament have less movement.

2. When one alters the tension of the filament, it becomes possible easily and accurately to regulate the sensitivity of the string galvanometer, and I am led to believe that the production of these curves satisfy the requirements of the International Committee for the Unification of Physiologic Methods. It requires little effort to understand that a certain number of millimeters of a deflection is comparable to the number of millivolts of a deflection of a corresponding degree of tension or to the number of micro-amperes of current.

3. With the string galvanometer, the deflection is exactly proportional to the strength of the current and maintains a complete equality between the deflections to the right and to the left, while with the capillary-electrometer, the proportionality between the deflections and the potential differences as it occurs with mercury and sulphuric acid, leaves much to be desired.

4. With the capillary-electrometer, the excursions of the meniscus become restricted by the friction of the mercury and the sulphuric acid owing to their passage through a small tube. Tiny invisible particles of dust may appreciably retard the movements of the mercury meniscus. Many capillary tubes must be replaced after relatively short usage, owing to the fact

that the movement of the meniscus ceases. Electro-magnetic suppression *(Dämpfung)* by chance can be influenced by variations in the strength of the field and conditions of the galvanometer.

5. The electrical isolation of the string galvanometer is more readily effected than is that of the capillary-electrometer, and a recession phenomenon does not occur in the galvanometer.

6. The filament offers certain advantages for observation in the projection of a clear-cut, magnified image. The aperture of the lens system, whereby the image is produced, is, as you know, of great importance in bringing about the necessary magnification. We can be certain that the use of a large aperture would be possible in the projection of the mercury meniscus of the capillary-electrometer, but it is evident that this offers no advantages as long as the illuminating lenses possess a smaller aperture. The illuminating lens in the capillary-electrometer is separated by the tubes or the chamber of sulphuric acid from the mercury; the focal distance of this lens thus must be relatively greater, its aperture small.[6] On the contrary, with the string-galvanometer, the distance from the filament can be shortened at will by manipulating the illuminating lens as well as the projection lens.[7] It is thus possible, with the dry system, to use a much larger aperture to good advantage.

One generally projects the image through a nearby effective cylindrical lens which concentrates the light on a photographic plate, perpendicularly to its axis. By this concentration of the light, the filament has a distinct advantage over the mercury meniscus, for, owing to the straight image of the filament, a sharp contrast between light and dark is maintained, while the curved mercury meniscus results in a less distinct outline.

7. And lastly, the operation of the string galvanometer is simpler. No special handling of the instrument is required when it is not in use. It is, thus, always ready for further use, which cannot be said of the capillary-electrometer.

On Plate VII the electrocardiograms of six persons are reproduced as registered by the string galvanometer. During the photography of the curves, the shadows are regularly projected in a co-ordinated manner on the sensitive plate, according to the accepted method of Garten.[8] The distance between the lines is arbitrarily chosen so that the entire field is divided into squares of about 1 sq. mm.

The speed of movement of the photographic plate conforms to 25 mm. per second, so that an abscissa of 1 mm. has a value of 0.04 second, while the tension of the filament is so adjusted, that an ordinate of 1 mm. corresponds to 10^{-4} volt of electromotive force. By the selection of these arbitrary values, the curves, nevertheless, meet the requirements of the International Committee.

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[6]One observes that under these circumstances a more opaque image is dealt with.
[7]At least, if one does not wish to sacrifice other important advantages for projection.
[8]Dr. Siegfried Garten, Ueber rhythmische elektrische Vorgänge im quergestreiften Skelett-muskel. Abhandl. der Kgl. sächs Gesellsch. der Wissenshaften zu Leipzig, math.-phys. Classe, Bd. 26, Nr. 5, S. 331, 1901.
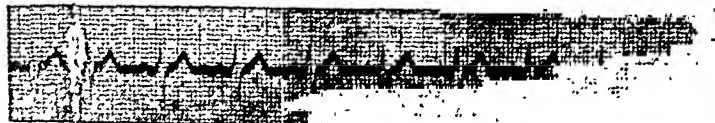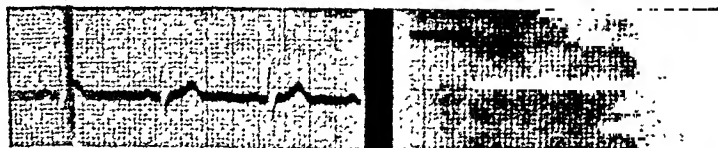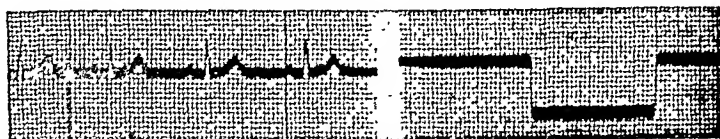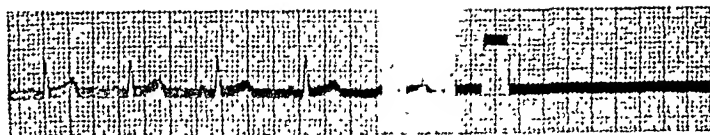
PLATE VII

Abscis. 1 mm. = 0.04 sec.   *Ordin.* 1 mm. = 10⁻⁴ volt.

At the outer end of most of the photograms, a normal curve is pictured, which is presented to show that the resistance of the human body is compensated for by the introduction of 1 millivolt of electromotive force into the circuit. In Nr. 6 the inscription of the normal curve is omitted, while in Nr. 4, twice 1 millivolt is utilized. This figure shows the perfect proportionality that exists between the deflections and the electromotive forces.

Although the magnification is 660 fold, it must not be expected that the edges of the filament will be sharply defined; nevertheless, one is able with certainty to detect a 0.1 mm. displacement of the edge of the filament. For that purpose if we only consider one of the normal curves, i.e., Nr. 4, one can be readily convinced that a deflection of 1 and 2 millivolts, an accuracy as close as 0.1, amounts to 10 and 20 mm.

The movement of the quartz filament is aperiodic and rapid, as can be observed in the normal curves, so that the electrocardiogram as recorded by the string galvanometer accurately expresses the variations of potential existing at the time of registration between the right and the left hand of the person. This usually holds true, without appreciable error, for the lower peaks, P, Q, S, and T. A correction must be made for the high and pointed peak, R, especially in photograms Nrs. 3 and 4, whereby the upstroke of the peak is moved a trifle to the left. However, the correction required still remains slight, and its value for the displacement to the left is approximately less than 0.2 mm.

If one desires greater accuracy, the curves of the string galvanometer may be treated in the same manner as those of the capillary-electrometer, whereby values are derived from the registered curve for the construction of a new curve. But this in many instances will be unnecessary.

The photogram Nr. 3 represents the electrocardiogram of the same person whose capillary-electrometer curve is pictured in the text figure. If one compares the registered curve Nr. 3 of the plate with the earlier constructed curve in Fig. 2 of the text, it becomes obvious that both are very similar. The peaks, P, Q, R, S, and T, not only are present in both curves, but both curves have relatively similar excursions. In the constructed curve, the distance between the ordinates represents 1 millivolt and that between the abscissae, 0.1 second; while in the galvanometric curve, the distance between the ordinates corresponds to 1 millivolt and the distance between the abscissae, to 0.04 second The galvanometric curve is consequently concentrated in the direction of the abscissae, a fact which becomes evident by casual examination of the curve.

The galvanometric curve, furthermore, gives the impression of symmetrical alterations from one peak to another, because the slight differences coincide better in the natural than in the constructed curve. According to the nature of the thing, only a limited number of points of the latter can be computed accurately, while the remaining portion of the curve must be constructed in such a manner as to connect, as nearly as possible, the points. However, the small differences are unimportant.

1904

LUDWIG ASCHOFF

DESCRIPTION OF RHEUMATIC MYOCARDITIS
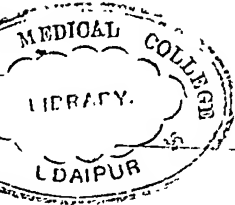
LUDWIG ASCHOFF

(Courtesy Journal of Pathology and Bacteriology

LUDWIG ASCHOFF

(1866-1942)

$\mathcal{L}$UDWIG ASCHOFF was born in Berlin on January 10, 1866, the son of a distinguished physician. He began the study of medicine at the University of Bonn, supplemented his training at the Universities of Berlin and Strasbourg and returned to Bonn, where he received his degree of Doctor of Medicine in 1889.

During his years as a medical student he had decided to become a pathologist and, therefore, shortly after he received his degree he accepted a position as assistant in pathology at the Pathological Institute of the University of Strasbourg, of which Friedrich von Recklinghausen (1833-1910) was director. Aschoff served under this great teacher for two and a half years. He then accepted a position as assistant to Johannes Orth (1847-1923) in the University of Gottingen, and soon become associate professor. After nine years he became ordinary professor of pathology at the University of Marburg. In 1906 he was appointed the director of the Pathological and Anatomical Institute in Freiburg im Breisgau, where he now resides.

The literature of pathology has been greatly enriched by Aschoff's many original contributions. He published as his dissertation an enlightening paper on the effects of the Staphylococcus pyogenes aureus on inflamed tissues. In 1900 he published, with Harvey Russell Gaylord (1872-1924), the "Course in Pathologic Histology." In 1902 his important study on Ehrlich's side-chain theory and its application to artificial immunization was published. From 1903 until 1909 Aschoff contributed many articles on the physiology and pathology of the heart. During this time he described, in collaboration with S. Tawara, the conductive system of the mammalian heart. He also published two more outstanding contributions which forever will be associated with his name. The first of these was his description of the rheumatic nodule, of which we print a translation; the second was his study of the double refractile substances.

In later years Aschoff elaborated on the pathology of the conductive system of the heart. A most interesting recent contribution on a phase of this subject was his paper[1] entitled "The Significance of the Conducting System in the Determination of Congenital Heart Lesions." This was read in 1937, when he was president of the International Association of Medical Museums.

The studies of Aschoff on the conductive system of the heart, on the double refractile substances, and his researches with Rinya Kawamura (1879-) on cholesterol metabolism finally led to an understanding of the pathologic processes in arteriosclerosis. This, in turn, led to his description of the reticuloendothelial system.

Aschoff's institute soon became the center of pathologic research and his ideas and contributions later in life are elaborated on in the many papers which he published in collaboration with his students. Among his students were many Japanese. The paper on the reaction of the reticuloendothelial system to dyestuffs, which he published with K. Kiyono, his researches in collaboration with Suzuki concerning the accumulation of vital stain substances in the kidney, and many other contributions—with

[1]Aschoff, Ludwig: The significance of the conducting system in the determination of congenital heart lesions, J. Tech. Methods 17: 95-96, 1937.

Skokichi Nagayo (1866-1910), Kusama, Mitsutano Ogata, Kawamura, and Tawara—are proofs of the great sphere of the influence of Aschoff which dominates Japanese pathology and medicine.

In later years Aschoff published his original contributions concerning the origin of the monocytes, the formation of gallstones, and the problems associated with the formation of bilirubin. His work on the pathology of the stomach, the pathogenesis of tuberculosis, and his studies concerning goiter are of particular significance.

In 1908, and later, in 1930, Aschoff made extensive investigations concerning the bacterial flora found in the normal and the inflamed appendix. In 1912 he published, in collaboration with B. von Beck, Oscar de la Camp, and Bruno Krönig, his important contribution on thrombosis.

The conception of general pathology, its correlation with physiology and clinical medicine, is probably best illustrated in his "Textbook of Pathologic Anatomy,"[2] first published in 1909. Today, after having appeared in several editions, it still is one of the most comprehensive and important surveys in its field.

In 1924 Aschoff published some of his contributions on pathology, including some of his American lectures,[3] delivered earlier in 1924. On June 29, 1938, he delivered the Finlayson Lecture[4] before the Royal Faculty of Physicians and Surgeons of Glasgow. The subject of his address was the history of the circulation. This was exceptionally well told.

In 1936, on the occasion of his seventieth birthday, Aschoff was the recipient of a tremendous ovation from his students and friends. Today he is justly looked on not only as the greatest living pathologist, whose name will ever be linked with his many original observations, but also as one of the most impressive personalities in the past and present history of medicine. *

[2]Aschoff, Ludwig., ed.: *Pathologische Anatomie. Ein Lehrbuch für Studierende und Ärzte*, bearbeitet von E. Albrecht (et al), Jena, G. Fischer, 1909, 2 v.

[3]Aschoff, Ludwig: *Lectures on Pathology*, New York, 1924, Paul B. Hoeber, 365 pp.

[4]Aschoff, L.: The History of the Circulation, Glasgow M. J. 130: 53-75, 1938.

*[Dr. Aschoff died in 1942.]

CONCERNING THE QUESTION OF MYOCARDITIS*

By

MR. L. ASCHOFF

Marburg

GENTLEMEN: Those who foster the myogenic theory of heart muscle contraction as originated by the Leipzig physiologic and clinical groups, also foster the present day teaching of myocarditis. The myogenic theory is supported by such good evidence that it occupies a firm, unassailable position in opposition to the neurogenic theory and pathology and pathologic anatomy is absolutely necessary in the consideration of this subject. The myogenic theory states that the automatic movement of the heart muscle lies wholly in the muscular tissue and that the existing nerve centers in the heart muscle, as well as those nerve paths leading to the heart, only serve to mediate reflexes from the heart to the rest of the vascular system and the entire organism, and in a reversed manner. Through these means the automatic movement of the heart can be strongly influenced, yes, be entirely stopped, but one could never be assured that the heart would be stimulated through nerve impulses or that its automaticity could be assured through its nerve centers. A marked alteration in the movement of the heart, even stand-still of the heart, can occur in two entirely different ways, through abnormal stimulation of the nerve innervation of the heart and through changes in the heart muscle itself. It is therefore necessary in every case of severe disturbance in the action of the heart to conduct a thorough investigation, both of the nervous system, that is, the centers in the medulla oblongata, the nerves, and heart ganglia, and of the heart muscle itself, in order to venture a safe opinion regarding the ultimate cause of the heart disturbance, if the heart and its nerve apparatus can be implicated as the origin of this disturbance and not the rest of the vascular system. A systematic investigation of the nerve apparatus is, however, confronted by great difficulties, as is the systematic investigation of the heart muscle according to the method inaugurated by Krehl, which, owing to its tediousness, was only carried out in a few definite forms of heart disturbances. Thereby, especially through the surprising results confirmed by Krehl and Romberg the weakness of the heart muscle following

the protracted course of scarlet fever, diphtheria, typhoid fever, *et cetera*, marked changes occur in the form of interstitial inflammation which appears to satisfactorily explain the heart injury rather than the necessity of reverting to disease of the nervous system. Likewise, the important question of why the heart muscle in valvular insufficiency ultimately fails in spite of its hypertrophy, was apparently completely solved by the Leipzig clinicians, through the demonstration of an interstitial myocarditis.

Plainly the myocarditis with valvular insufficiency appeared to be the best evidence for the myogenic theory of automatic contraction of the heart muscle, and in this connection, changes in the heart nerves and ganglia are hardly to be expected and are not disclosed, and the interstitial changes appear sufficiently widespread to explain the weak and irregular contraction of the heart. Is this actually so? I will not fail to mention that this observation was not confirmed by other investigators. And when one studies the work of the Leipzig school more closely, one finds that interstitial myocarditis is only accepted in a part of the cases, and in these, often to a small extent. Further proof seemed desirable. I had Dr. Tawara examine 150 hearts without special selection; of these, sixty were examined in a manner approaching the systematic form of the method of Krehl. Among the latter were three cases of acute nonulcerating endocarditis, and eight cases of chronic endocarditis with marked stenosis and insufficiency of the aortic and mitral orifices. In addition, there was one case of pure acute myocarditis. All these cases clearly belong to the group of rheumatic diseases. In five cases, articular rheumatism had certainly occurred. It is only regarding those cases with myocardial changes associated with valvular insufficiency that I will briefly discuss here. The very time-consuming investigation of the remaining hearts is not yet completed.

Of these, two hearts hardened in formalin were selected, and sections obtained from the apex, the anterior and posterior papillary muscles, the posterior wall of the left ventricle, the septum, the papillary muscles, and the conus of the right ventricle, the right and left auricle, and the sections were stained by the various staining methods (van Gieson, Weigert's stain for elastic fibers, polychromic methylene blue, pyronine, incidentally, also according to the Gram method). In addition, corresponding areas were investigated by frozen sections stained for fat with hematoxylin-Sudan.

We were enabled through this comprehensive and somewhat tedious method to recognize all the changes in the heart substance, the muscle fibers, likewise the fibrous tissue and elastic fibers, the cellular elements of the connective tissue in their form and distribution. The result of this investigation is primarily a confirmation of the views of the Leipzig

school regarding the general occurrence of interstitial change in the heart muscle associated with valvular insufficiency. It, however, broadens the concept of the Leipzig followers, insofar as it permits us to more accurately establish the historic coordination of the products of inflammation, and thereby to find peculiar nodules, which appear to be specific for rheumatic myocarditis. These nodules were plentiful and clearly delineated in only two cases of recurrent endocarditis, but corresponded exactly in their location to the cellular growth in the other cases. They regularly occur in the neighborhood of small or medium-sized vessels, and most frequently were present in the vicinity of the adventitia. Or there existed simultaneously a disease of all the vascular layers, such as is described in arteritis nodosa. The aforementioned nodules are unusually small, mostly submiliary, and originate by the conglomeration of large elements, with one or more abnormally large indented or polymorphic nuclei. The arrangement of the cells frequently occurs in the form of a fan or a rosette. The periphery is formed by the large nuclei, the center by the paler or colorless appearing necrotic mass of confluent cell protoplasm. By cursory examination, the fan formations slightly resemble the necrosis of gout with a peripheral cell mantle, as is so frequently observed in the gouty kidney. The rheumatic nodules are not to be confused with tubercles or foreign body cells with more uniformly formed nuclei, but are of a configuration that more nearly resembles the larger nuclear elements in certain sarcomas or the infiltrations in pseudoleukemia. In all events, the nodules do not exclusively consist of such large nucleated cells, but also small and large lymphocytes, and polymorphonuclear leucocytes force themselves a short distance between the large cells of the periphery, or form a peripheral zone, and from there, irregular projections may extend far into the connective tissue partitions. In these richly cellular projections are found isolated cells with large nuclei, with all the transitions to a simple large leukocytoid element, which are even found in a normal manner in the neighborhood of the smallest vessels and appear very distinct in all inflammations. These leukocytoid elements are the large cells already described by Hayem and Romberg, the genesis of which, however, remains uncertain to them. From these large cells, which are the inflamed swollen adventitial cells of the vessels, the giant cell-like large nucleated element arises; these appear singly or are collected in nodules, and give the rheumatic cellular infiltration its peculiar configuration. It may be further stated that the number of eosinophilic nucleated cells in these nodules is extremely small. While in the one case the structure of the nodule gives the impression of a fresh cellular infiltration, in another case a partial or complete fibrous replacement of the nodule is evident. I have already remarked that this nodule formation shows an identical localization and proves its close relationship to that of the interstitial inflammatory

changes in other cases. Likewise, the nodule-free cellular infiltration in other cases of valvular insufficiency may be histologically recognized by its similar conformity, insofar as the large adventitial cells are concerned, in intimate mixture with large and small lymphocytes and lobulated nuclei; in part, the eosinophilic nucleated leukocytes contribute to the perivascular collection.

I investigated this peculiar nodular formation in somewhat more detail, and I found a very similar formation in a case of acute interstitial myocarditis in which no trace of acute or chronic endocarditis existed and except for visible skin hemorrhages, no visceral changes were found. The illness, three weeks after a healed hand injury, led to sudden death. The pattern of this myocarditis is almost identical to that in the case described by Askanazy in a Königsberger dissertation. Insignificantly small areas of necrosis were formed in the heart muscle fibers, pronounced punctate and cordlike cellular infiltration of the entire heart substance, predominated by eosinophilic nucleated leukocytes, and great numbers of Charcot-Leyden crystals in the rich, cellular, partly necrotic areas. In this case, in addition to the eosinophilic nucleated leucocytes, it is easily understood that the infiltrating adventitial cells also play an important role. Furthermore, isolated plasma cells, large and small lymphocytes, and also occasional fibroblasts, were found. But noteworthy of mention were the sparsely developed large cell nodules similar to those previously described. In this case, in which sepsis was presumed to have existed, all investigations for the demonstration of microorganisms were failures, in blood cultures from the vessels *intra vitam* from the heart during postmortem examination and in the stained sections. I believe, therefore, that in this case we are concerned with an infection similar to that occurring in rheumatic endocarditis, the causative agent of which is difficult to determine. Experiences during my stay in Göttingen have shown me that such cases of acute interstitial myocarditis, with a preponderant proportion of eosinophilic nucleated leukocytes and evident Charcot-Leyden crystals, are not so uncommon with and without existent valvular insufficiency. The condition is frequently linked with trauma, overexertion, contusion, and injury to the outer layer of the skin. How the entrance or the localization of the specific virus ensues, is not entirely clear. As we have the large cell nodule formation only in rheumatic endocarditis, and never in the typhoid heart, diphtheria heart, *et cetera*, we believe it permissible to conclude that it is especially characteristic of rheumatic myocarditis. On the other hand, the investigations of Dr. Tawara have necessitated restrictions in the teachings of the Leipzig school. It was, namely, in spite of the comprehensive systematic investigations, that the described interstitial changes in the other cases were so slight, that the symptoms of heart failure observed clinically could not be associated with the changes in the connective tissue struc-

ture. In five cases a connective tissue infiltration was virtually absent; in three cases only old, healed scars were found. Here, all important sections of the heart were examined; thus, in these cases interstitial myocarditis cannot be the cause of the heart failure, which was very pronounced in just two cases in which clinical data were available (the material came largely from the outside).

According to our experience, certain allowances must be made for those cases with valvular insufficiency, for the diminished efficiency of the heart muscle, at least in a large number of cases, may be sought for elsewhere than in progressive connective proliferation. This can only be sought in the muscle substance of the heart, or, if one does not wish to accept the myogenic theory, in the ganglia. Therefore, in all the cases, Dr. Tawara took sections through the left auricle in such a manner that larger and smaller ganglion cell groups were accessible and included in the investigation.

To consider first the ganglion cells: in spite of staining with polychromic methylene blue or pyronine, apparent changes were so seldom detected, and particularly in cases of pronounced heart failure, that the source of the muscle insufficiency must originate elsewhere. The tigroid substance was chiefly well maintained; likewise the nuclei and the nucleoli; only seldom was early disintegration of the chromatin substance evident, which could be interpreted as occurring after death. The occasional marked swelling of the basket cells was striking. In every case, fresh inflammatory or older scars were not detected in the connective tissue of the ganglia, as would be anticipated in the course of subacute or chronic inflammation of the heart valves.

There remained, then, only the muscular substance. Its significance was revealed by the comprehensive, outstanding, assiduous monograph of Albrecht, which appeared during our investigation. He considers the universal enlargement of the heart muscle and the ensuing degeneration the same as a parenchymatous inflammation, and reiterates the view that the diminution in the functional activity of the hypertrophied heart muscle can be explained in a different manner from that expressed by the Leipzig school. He based his views chiefly on the fact, that with the evident hypertrophy, no increase in the effective contraction substance occurred; it was not in the fibrils, but only in the sarcoplasm, that the nuclei assumed the form of "Leistenkerne" already described by Romberg; that these progressive changes result in a regressive form of granular degeneration of the protoplasm, a shrinkage or swelling of the nuclei, until finally, instead of single muscle cells, only nuclei-containing pigmented collections of protoplasm alone remain. The connective tissue infiltration is somewhat secondary.

Unfortunately, the investigations of Dr. Tawara were in no manner able to support these findings. Without regard to Albrecht's basic error

that conjoined muscle cells actually exist while all these striations only represent differences in form and condition of special contraction phenomena, it is easy to conclude that in the hypertrophied fiber segments, in addition to the increase of sarcoplasm, there also occurs thickening of the fibrils; that the peculiar elevated nuclear formations of Romberg are not pathologic, but already occur early in youth, or at least exist in the physiologic *Anlage*, but only appear more distinctly in the hypertrophied muscle bundles. All of Albrecht's depicted nuclear formations are also found in hearts in all decades which are not hypertrophied, except for the fact that the nuclei are smaller and the formation is less distinctly seen. The observation that the nuclei of the heart of a youth or an adult under normal circumstances occur in oval or rodlike formations, with smooth surfaces, must finally be relegated. They only exist in this condition in the newborn and young child. Even in an older child, more so in an adult, the nucleus assumes a flat form on both sides, which like a flat ring is bent over the surface with points and extensive projections. The greatest diameter of the nucleus is frequently at right angles to the longitudinal direction of the fibers. The size of the nucleus and its chromatin content are very variable. Definite degenerative nuclear changes do not exist, not considering the disintegration of the nuclear substance. One can only distinguish between simple, hypertrophic or atrophic nuclei; but, nevertheless, all transitions are present and the ranges are difficult to determine.

On the other side, concerning the views brought to the foreground by Albrecht regarding the changes of the nuclear degeneration of the muscle fibers, which occur independently of embolic processes or inflammatory necrosis, it may be said that we have not observed these changes. Vacuolar nuclear degeneration and fatty infiltration also play a large role in hypertrophied hearts, and surely impair heart efficiency, but do not lead to an appreciable degree of loss in muscular substance. But they can also exist in hypertrophied hearts that lag in their work, fail, or possess such slight reserve that it does not suffice for the explanation of the heart failure. I surely do not wish to consider the purely agonal occurrence of fragmentation if I do not consider pigmentation, the beginning of which may already be found in the first decade of life.

When, therefore, we previously could find no histologic pattern for the failure of the muscle mass, it was necessary to consider that not the muscle mass as such, but only a certain part, was injured, namely; the so-called atrioventricular bundle, which gives automaticity according to the myogenic theory. On that account, Dr. Tawara studied serial sections from the region of the membranous portion, together with parts of the auricular and ventricular septa in a series of eight cases, of which three belonged to the above-mentioned category. He was enabled to prove that the atrioventricular bundle in all cases corresponded to the

descriptions of Kent, His, and Retzer. Contrary to Retzer's findings, it was apparent that in the newborn as well as in the adult, a marked difference existed between the strength of the fibers and the form of the nuclei in the fibers of the atrioventricular bundle and the ventricular muscle. At its entrance in the auricular septum, the atrioventricular bundle forms a dense texture in the shape of a node. The result of the investigation was that in no case, as in those three cases of rheumatic endocarditis that are of special interest to us in this consideration, were appreciable changes discovered.

Therefore, on the grounds of Dr. Tawara's investigation we must conclude: (1) that the enlargement of the heart muscle in valvular insufficiency produces a true hypertrophy, and (2) that inflammatory changes do not have the described significance and do not explain the decrease in efficiency of the hypertrophied muscle, but that the heart muscle weakens and we are unable to observe by means of our present-day laboratory aids, degenerative changes of a greater extent in the muscular substance. And a circumscribed lesion of the atrioventricular bundles does not enter into the consideration of the cases thus far studied.
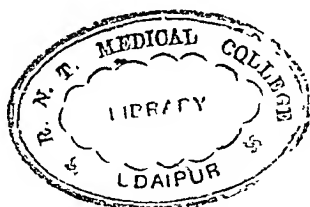
1907

ARTHUR KEITH AND MARTIN FLACK

DEMONSTRATION OF THE SINOAURICULAR NODE
(NODE OF KEITH AND FLACK)

SIR ARTHUR KEITH

(Courtesy Journal of Anatomy.)

SIR ARTHUR KEITH

(1866-1955)

SIR ARTHUR KEITH, senior author of the classic we are reproducing, was born in Old Machar, Aberdeen, Scotland, on February 5, 1866. He is the son of John and Jessie Macpherson Keith.

Arthur Keith studied at the University of Aberdeen, where he received the degrees of Bachelor of Medicine and Master of Surgery, with first class honors, in 1888. In 1894 he received the degree of Doctor of Medicine, with highest honors, from the same university. In 1894 he also qualified for membership and received his fellowship in the Royal College of Surgeons. He also studied at University College, London, and the University of Leipzig.

In 1899, Keith married Cecilia, daughter of Tom Gray, the artist. She died in 1934.

Early in his career as an anatomist, Keith developed a profound interest in anthropology. In 1896 appeared his first work, entitled "An Introduction to the Study of the Anthropoid Apes." Other works by Keith about anthropology are: "Ancient Types of Man" (1911), "The Antiquity of Man" (1915; second edition, 1925), "Nationality and Race from an Anthropologist's Point of View" (1919), "Concerning Man's Origin" (1927), and "New Discoveries Relating to the Antiquity of Man" (1931).

Keith also contributed two books on Darwinism: "Religion of a Darwinist" (1925) and "Darwinism and Its Critics" (1935). The work for which he is probably most widely known is his "Human Embryology and Morphology." This was first published in 1901, and the fifth edition of this work appeared in 1933. He also is author of "The Human Body" (1912) and "Engines of the Human Body" (second edition, 1925).

To the field of orthopedics, Keith contributed an important historical work entitled: "Menders of the Maimed" (1919). In this volume he traced the development of the anatomic and physiologic principles underlying the treatment of injuries to muscles, nerves, bones, and joints. At the time of the publication of this work, Keith was conservator of the museum and Hunterian professor of the Royal College of Surgeons of England. He was appointed to this position in 1908 and continued in it until his retirement in 1933. Before this time, and at the time he and Flack were working on the node, Keith was lecturer in anatomy, in London Hospital Medical College.

In 1921 Keith was created a baronet. He had served as secretary of the Anatomical Society of Great Britain from 1899 to 1902, and as president from 1913 to 1917. When, in 1916, the "Journal of Anatomy" became the property and official organ of the Anatomical Society, he was appointed acting editor. He held this position for seventeen years and the unqualified success of this authoritative journal is the result, to a great extent, of Sir Arthur's indefatigable labor and skillful guidance.

Sir Arthur has held many enviable positions. In 1913, he was elected a member of the Société d'Anthropologie de Paris. He served as Fullerian professor of physiology at the Royal Institution from 1917 to 1923. He was secretary of the Royal

[743]

Institution from 1917 to 1923, and in 1926 and 1927 was treasurer. From 1930 to 1933 he was rector of the University of Aberdeen. He has received the honorary degree of Doctor of Laws from the Universities of Aberdeen, Birmingham, and Leeds. In 1920 he received the degree of Doctor of Science from the University of Durham. In 1923 he received the same degree from the University of Manchester and in 1930 the University of Oxford gave him another. He is also a fellow of the Royal Society of London.

At present, Sir Arthur is master of the Buckston Browne Research Farm in Downe, Farmsborough, Kent, England.* It was not far from that farm, near Flack's native village of Borden, in a small cottage in an orchard, that Keith and Flack, during their college vacation, carried out their valuable study on the node.[1]

MARTIN WILLIAM FLACK

(1882-1931)

*M*ARTIN WILLIAM FLACK was born in the village of Borden in Kent, England, in 1882. He received his preliminary education at the Great Yarmouth and Maidstone Grammar Schools. Later he matriculated at Keble College, Oxford, where he studied general science as well as medicine. Among his teachers were Arthur Thomson (1858-1935), Francis Gotch (1853-1913), and John Scott Haldane (1860-1936). In 1905, Flack received the degree of Bachelor of Arts from Oxford University.

Flack completed his medical studies at the London Hospital, where he had been awarded the Price Scholarship. There he came under the direction of Sir Leonard Erskine Hill (1866-   ), who then was a lecturer in physiology, and Sir Arthur Keith, who was a lecturer in anatomy, in the London Hospital Medical College. Arthur Keith[2] at that time was studying the nervous and muscular structures of the heart and invited young Flack to work with him.

In 1906, Tawara[3] announced his discovery of a system of conducting musculature between the auricles and ventricles of the mammalian heart. Keith and Flack verified the truth of Tawara's discovery and set out to trace the evolution of the auriculoventricular connecting system. They also desired to ascertain if, in the region wherein the beat of the heart was believed to begin—namely, at the termination of the superior vena cava in the right auricle—there existed tissue of the nature of the node which Tawara discovered at the beginning of the auriculoventricular bundle. They found in all mammalian hearts, at the expected site, a collection of peculiar muscular tissue. This tissue, they named the sinoauricular node, inferring from its position, and from its resemblance to the tissue at the beginning of the ventricular bundle, that this node was concerned in the inception of the heart beat. This important classic, published in 1907 in the ''Journal of Anatomy,'' we are happy to reproduce for our readers. At a later date, according to Keith, Lewis demonstrated the exact site at which the normal heart beat originates and proved that the site coincides with the position of the sinoauricular node.

Flack at the time of his clinical studies at London Hospital acted as assistant demonstrator of physiology under Sir Leonard Hill. He assisted Hill in research problems which were mainly concerned with circulation and respiration. This experience was of great benefit to him later, when he studied the medical problems of aviation.

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[1]Personal communication from Sir Arthur Keith.
[2]See preceding biographic sketch.
[3]Tawara, S.: *Das Reizleitungssystem des Säugethierherzens*, Jena, 1906, Gustav Fischer.
*[Sir Arthur Keith died in 1955.] [744]

MARTIN FLACK

Photograph by Russell and Sons

In 1908, Flack received the degrees of Bachelor of Medicine, Bachelor of Surgery, and Master of Arts from Oxford University. That same year he was married to his cousin, Cecile Cooper. Four children, two sons and two daughters, were born to them.

In 1909, Flack received a Radcliffe Travelling Fellowship. This gave him the opportunity of studying on the Continent, study for which he had long had a desire. At Bern, Switzerland, he worked under Hugo Kronecker (1839-1914) and Asher, studying the nerve supply to the thyroid gland. He also spent some time at Liège, Belgium, where he studied the heart under the great Léon Frédéricq (1851-1935), who had been chosen by Théodor Schwann (1810-1882) to become professor of physiology in the University of Liège in 1881 at the age of thirty, and who proved the existence of fibrinogen in the blood plasma.

Flack returned to London and continued as demonstrator in physiology in Hill's department at the London Hospital. He collaborated with Hill in many investigations and later, in 1909, became co-author with Hill of the work: ''A Textbook of Physiology.''

In 1913, Hill was asked by the Medical Research Council to establish a department of applied physiology. He chose Flack to assist him in this undertaking. The World War began the next year and in the early part of the struggle, Flack lent his services to the army. With Mervyn Gordon he combated the epidemics of cerebrospinal fever then current. As a result of this service he was breveted lieutenant colonel in the Royal Army Medical Corps, and on the formation of the Royal Air Force Medical Service he was made wing commander and director of medical research.

The World War demonstrated the need for the investigation of the oxygen starvation of airplane pilots at high altitudes, and Flack was chosen for this work. He not only made an inquiry into the need of oxygen by flyers at high altitudes, but also developed a series of comprehensive tests to determine the physical fitness of applicants for the air service. The results of these important studies were made the subject of his Milroy Lectures, which he delivered before the Royal College of Physicians in 1921. In 1923 Flack was promoted to the rank of group-captain.

Group-Captain Flack represented the British Medical Services at the San Antonio meeting of the Association of Military Surgeons in 1924. He also attended the meeting of this organization in Washington, D. C., in 1930.

Flack suffered for many years from a rheumatic affection of the heart but in spite of this physical handicap achieved great ends. His death on August 16, 1931, was the result of a septicemia.

THE FORM AND NATURE OF THE MUSCULAR CONNECTIONS BETWEEN THE PRIMARY DIVISIONS OF THE VERTEBRATE HEART*

By

ARTHUR KEITH, M.D. (Aberd.), F.R.C.S. (Eng.)

Lecturer in Anatomy, London Hospital Medical College

And

MARTIN FLACK, B.A. (Oxon.)

Assistant Demonstrator of Physiology, London Hospital Medical College

Introductory.—In a paper contributed to *The Lancet* some months ago (5), the writers confirmed and extended the discovery of Tawara (7), viz. that there is within the mammalian heart a system of peculiar musculature (the a.-v. system) which, beginning as a small root (the *Knoten*) in or near the base of the interauricular septum on the right side, eventually spreads out in an arborescent form beneath the endocardium of both ventricles, its final twigs becoming everywhere continuous with the ordinary musculature of the ventricles. It seemed to us essential to examine other regions of the heart for such peculiar musculature. Moreover Wenckebach (8) has demonstrated by exact clinical methods that a delay may occur in the conduction of the cardiac impulse from sinus to auricle. This fact stimulated us to investigate fully the nature of the muscular connection between the sinus and the auricle, which has already been partly described by Wenckebach in the human heart.

In this paper therefore the writers propose to deal with the results of an extended inquiry, made with three objects in view:—

I. To ascertain the extent, nature, and position of the muscular connection or connections between the primary divisions of the heart in all classes of the vertebrate kingdom.

II. To seek in the sinus, auricle, and bulbus cordis for a differentiation in form and structure of a system of muscle fibres corresponding to that now known to exist in the ventricle: in short, to ascertain whether the musculature in which the heart-impulse is held to arise, and by which it is conducted, differs in form and structure from that which is mainly contractile in nature.

III. To trace the evolution of the a.-v. muscular system, as found in the human heart, from the simpler and more definite form seen in the heart of fishes.

Material.—It is important that those who may consult this paper should know the exact material used by us in this inquiry, and its method of preparation. In the appended list of material we do not include the numerous hearts which have been dissected by knife and forceps, but only those which have been examined by a series of microscopic sections.

List of Material

Fishes.—Eel, dog-fish, salmon.

Amphibia.—Frogs (3).

Reptilia.—Lizard (species unknown), tortoise, turtle.

Birds.—Sparrows (2), goldfinch.

Mammals (other than human).—Mole, porpoise, dolphin, kangaroo, wallaby, whale (*B. musculus*), mouse, shrew-mouse, rat (2), kitten (2), ram, pig, cart-horse, pony, foetal gibbon.

Human.—Embryos (2), normal hearts, malformed hearts, and fifteen hearts having definite pathological lesions.

Method of Preparation.—For macroscopic specimens for dissection a modification of Kaiserling's method was used. The great advantage of this method is that the natural colour of the muscle fibres returns after fixation, thereby rendering easier the dissection of the different systems of muscle fibres. The procedure is as follows:—

(1) The heart must be well washed in running water for 12 hours prior to fixation, and the cavities stuffed with tow or cotton-wool.

(2) The specimen is then fixed in the following solution:

Formalin, 200 c.c.
Water, 1000 c.c.
Potassium nitrate, 15 grams.
Potassium acetate, 30 grams.

In this solution it remains at least 24 hours, and longer if it be large, hard, or tough.

(3) Specimen placed in 80 per cent spirit until its colour returns.

(4) Kept in equal parts of glycerine and water.

In the preparation of microscopic specimens stages (3) and (4) are omitted. After (2) the specimen, or the desired parts of it, is well washed in running water. It is then transferred to spirit (24 hours), next alcohol in stages from 70-100 per cent. for 24-48 hours, then in xylol until clear, and finally embedded in paraffin. We have found that the process of embedding is much facilitated by exhausting the incubator. By this means clear, firm blocks with no trace of air-bubbles are obtained. The blocks were cut in the main at 10μ, except when it was desired to study the minutest structure of the specimen, when they were cut from 4μ to 7μ in thickness, according as the nature of the tissue permitted. The

sections were stained by Ehrlich's acid haematoxylin and Van Gieson's stain, dehydrated, and mounted in Canada balsam. It is important to overstain with haematoxylin, otherwise the nuclei will not be well seen, owing to the decolorising action of the second stain.

Literature.—With regard to literature, we have been unable to find any previous paper approaching the nature of our research. The writings of Gaskell (2), MacWilliam (6), and Engelmann (1) have proved of great service. We accept the teaching (1) that the heart's impulse is conducted by the cardiac muscle tissue, (2) that normally the impulse arises in the musculature of the sinus, setting the heart's rhythm, and then passes to the auricle and ventricle, finally reaching the bulbus cordis.
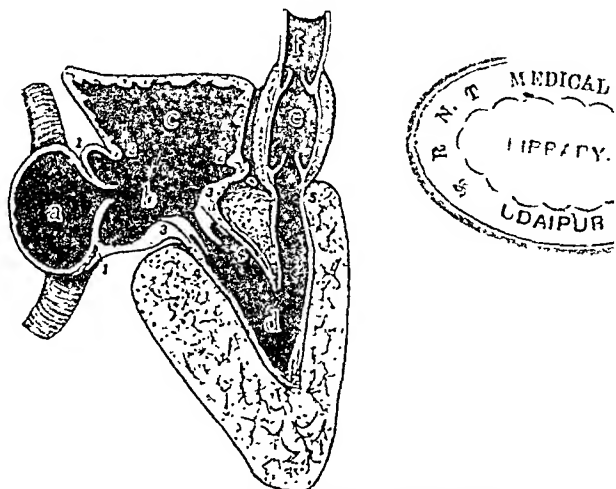
Fig. 1 —Diagram of a generalised type of vertebrate heart—combining features found in the eel, dog-fish, and frog—showing the primary cardiac chambers and their lines of union.

a, sinus venosus and veins; *b*, auricular canal; *c*, auricle; *d*, ventricle; *e*, bulbus cordis; *f*, aorta; 1-1, sino-canalar junction and venous valves; 2-2, canalo-auricular junction; 3-3, annular part of auricle, containing special muscle fibres; 4-4, invaginated part of auricle; 5, bulbo-ventricular junction. By the longitudinal fibres lining the ventricle there is a connection between the annular fibres of the auricle and the bulbus musculature.

The Primary Divisions of the Vertebrate Heart.—Before proceeding to describe the muscular connections between the primary divisions of the heart, it is necessary for us to define exactly what we regard as such. They are well seen in the generalised diagram (Fig. 1). There are five primary divisions of the heart:—

    (1) The sinus venosus (*a*).
    (2) The auricular canal (*b*).
    (3) The auricle (*c*).
    (4) The ventricle (*d*).
    (5) The bulbus cordis (*e*).

There are four junctional lines:—

(1) *The Sino-canalar*, marked by the venous valves, the free margin of the valves forming the boundary-line between the cavity of the sinus and cavity of the auricular canal (*1-1* in Fig. 1).

(2) *The Canalo-auricular*, marked by a thickening of the musculature round the ostium of the auricle, situated on the dorso-lateral wall of the auricular canal (*2-2* in Fig. 1).

(3) *The Canalo-ventricular*, marked in the mammalian heart by the auriculo-ventricular valves, the free margins of which separate the cavity of the auricular canal from that of the ventricle (*4-4* in Fig. 1).

(4) *The Bulbo-ventricular*, situated at the junction of the ventricle and bulbus (*5* in Fig. 1).
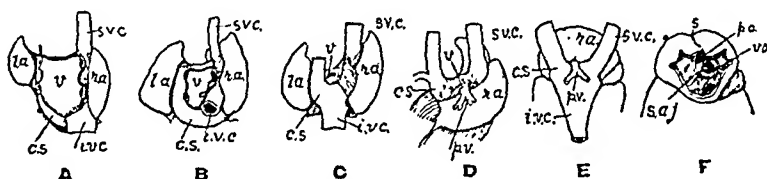
FIG. 2.—Series of diagrams to illustrate the parts in the human heart corresponding to the sinus of the primitive heart.

A, dorsal view of auricular part of the human heart; B, corresponding view of wallaby's heart; C, corresponding view of the heart of a child in which the lungs were fused and the vestibule of the left auricle consequently unexpanded; D, corresponding view of the heart of a malformed fœtus in which the inferior vena cava was absent; E, corresponding view of the heart of the frog (Gaupp); F, corresponding view of the heart of the frog, the interior of the sinus venosus being exposed to show that the pulmonary veins open within the sino-auricular junction. In this figure, *p.o.*, orifice of pulmonary veins; *v o.*, orifice of sinus venosus; *s*, attachment of interauricular septum; *s.a.j.*, sino-auricular junction. *v.*, vestibule; *r.a.*, right auricle; *l a.*, left auricle; *s.v.c.*, superior vena cava; *c.s.*, coronary sinus; *i.v c.*, inferior vena cava.

The Primary Divisions and Junctional Lines in the Mammalian Heart.— Our knowledge of the heart has been derived in great part from experiments made on the simpler hearts of the eel, frog, and turtle; in order to transfer accurately that knowledge to the mammalian, and more especially to the human heart, it is necessary to identify in them the primary divisions which are seen so clearly in the simpler hearts. We propose, therefore, in the first place, to identify the five primary divisions above mentioned in the human heart.

The Sinus Venosus of the Human Heart.—In Fig. 2 is shown a series of illustrations of views of the sinus venosus in various hearts; the view represents the venous or auricular end of the heart, looked at from the dorsal side. The sinus (see *E*) is formed by the union of three great vessels—the right duct of Cuvier (*rt. sup. v.c.*), the left duct of Cuvier (*lft. sup. v.c.*), and the inferior vena cava (the hepatic vein of fishes). In the human and in the mammalian heart, the musculature of the auricular canal has grown over and submerged the greater part of the sinus (*o*, Fig. 3); two parts only are left exposed on the surface of the heart—(1) the musculature of

the superior vena cava, (2) the musculature of the coronary sinus (the representative of the left superior vena cava) (see Fig. 2 *A, B C*). But if a section be made across the line at which the sinus becomes submerged (the stria terminalis of His), a second or deep stratum of musculature is seen (beneath *o*, Fig. 3): this probably belongs to the sinus venosus, since it extends beneath the endocardium of the auricle, from the position of one venous valve to that of the other. Besides these three definite remnants of the sinus musculature, there is also the musculature—or part of it—in the Thebesian and Eustachian valves, these being remnants of the right venous valve. There is often also to be found a thin muscular layer along the lower border of the fossa ovalis: it occupies the position of the left
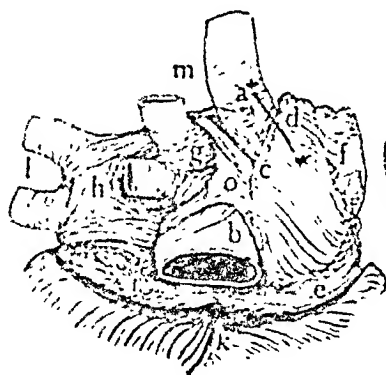
FIG. 3.—The auricular part of the human heart from behind, showing the musculature of the termination of the great veins.

a, superior vena cava surrounded by musculature derived from the sinus; *b*, inferior vena cava; *c*, to the right of the sulcus terminalis, above *c*, sinus fibres cross the sulcus to join auricle proper; *d*, at sino-auricular junction, where peculiar musculature is found most abundantly; *e*, annular fibres of auricle; *f*, appendix, *g*, fibres passing from interauricular septum to vestibule of left auricle between the two left pulmonary veins, *h*, vestibule; *i*, coronary sinus, showing continuity of fibres with right and left auricles; *k*, base of ventricles at interventricular sulcus; *l*, left pulmonary veins; *m*, constant band passing from above musculature to vestibule of left auricle; *o*, fascicle of auricular canal submerging sinus; ** represent line of section of Fig. 6, A

venous valve, and is probably derived from it. Thus, the chief remnants of the sinus venosus have to be sought for in the right auricle of the human heart. Its musculature is represented by:—

(1) The termination of the superior vena cava.
(2) The coronary sinus.
(3) The submerged stratum.
(4) The remnants of the venous valves.

It may be, however, that there are also remnants of the sinus in the left auricle of the human heart. In Fig. 2, *F*, it is seen that in the heart of the frog the musculature of the sinus at the sino-auricular junction (*s.a.j.*) includes within it the orifice of the pulmonary veins. This is also seen in the heart of the malformed foetus, Fig. 2, *D*. It is possible, therefore,

that, as the part of the auricular canal (v.) which is to become the vestibule of the left auricle expands, a part of this sinus musculature is also involved in the process, and may persist in the left auricle of the human heart around the orifices of the pulmonary veins.

In a part or in all of this sinus musculature the heart rhythm is believed to be initiated.

The Auricular Canal of the Human Heart.—In the simplest form of heart the auricular canal, which joins the sinus venosus to the ventricle, is differentiated into three parts (see Fig. 1)—(i) a basal part (opposite the auricle), (ii) an annular part (3-3), (iii) an invaginated or intraventricular part (4-4). The invaginated part forms an isolated layer beneath the auriculo-ventricular valves, its musculature becoming continuous with that of the ventricles near the apices of the valves (Fig. 1). Only a small part of this musculature remains in the human heart; it forms the a.-v.
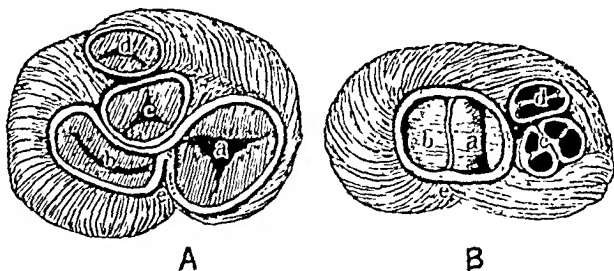
Fig. 4.—To illustrate the infolding and modification of the auricular ring in the mammalian heart.

A, base of the ventricles of human heart; B, base of the ventricles of heart of turtle. *a*, right auriculo-ventricular orifice; *b*, left auriculo-ventricular orifice; *c*, orifice of aorta; *d*, orifice of pulmonary artery; *e*, posterior part of the auricular ring, which becomes infolded in the mammalian heart. The stippled part near *e* represents the only part which remains undifferentiated in the mammalian heart.

bundle. This we shall treat of more fully later on. The annular part of the canal—the "auricular ring," as we shall term it in this article—has in the human heart become submerged in the auriculo-ventricular groove just above the base of the auriculo-ventricular valves (c, Fig. 3). Only one essential change has taken place. This can best be clearly explained by the help of a figure.

In the reptilian as in the amphibian heart (Fig. 4, B) the annular part forms a simple ring; the interauricular septum lies within it, separating the right from the left a.-v. orifice. But in the mammalian heart the simple annular form has been lost; owing to the extension of the bases of the ventricles backwards under the basal wall of the auricular canal, the annular part has been folded as shown in Fig. 4, A, so that the mesial folded part has now come to rest upon the upper or auricular margin of the interventricular septum. From this superventricular fold of the annular ring begins the a.-v. bundle (stippled in Fig. 4, A).

The basal part of the auricular canal is best defined by explaining its origin. The auricle or auricles are outgrowths from the dorsal wall of the auricular canal (see Fig. 1); the ventral wall remains unspecialised as the basal part. The basal part, it will be seen, is continuous with the sinus venosus, with the ostium of the auricle, and with the auricular ring. From a physiological point of view the basal part of the auricular canal is most important, since both Gaskell and MacWilliam found that it was a path of conduction from the sinus to the ventricle, so that a sino-ventricular rhythm could occur. It is therefore interesting to see whether the possibility of this rhythm remains in the mammalian heart. In the human, as in the mammalian heart, the basal wall has become profoundly modified by two great cardiac transformations which have occurred with the evolution of the pulmonary system. These changes are: (1) the formation of the interauricular septum, (2) the formation of a vestibule to the left auricle (Figs. 2 and 3). The basal wall has supplied a large part, if not the whole, of these two structures. As the fibres of the lowest part of the interauricular septum come into intimate relation with the annular ring, it will be seen that it is therefore possible for a sino-ventricular rhythm to occur in the human heart. Indeed, a layer of longitudinal muscle fibres passes directly from the superior vena cava into the auricular septum, and thus reaches the musculature from which the a.-v. bundle commences. The musculature of the three parts of the auricular canal is represented in the human heart thus:—

(1) The basal part by the interauricular septum and by the vestibule of the left auricle.

(2) The annular part by the circular fibres surrounding the ostia above the bases of the auriculo-ventricular valves. The annular fibres also descend for some distance on the septal cusp of the tricuspid valve.

(3) The invaginated part of the a.-v. bundle.

The Auricles of the Human Heart.—In the fish's heart, the common auricle forms a well-demarcated outgrowth on the dorsal wall of the auricular canal. The ostium, by which it opens on the canal, is surrounded and indicated by a thick circular ring of musculature (Fig. 1, 2-2). In the mammalian heart, the development of the interauricular septum and of the vestibule of the left auricle from the basal wall has led to a division of the auricle and to a wide separation of its two parts (see Fig. 3). However, in the mammalian, and especially in the human heart, a prominent ridge of musculature, commencing in the right auricle immediately in front of the termination of the superior vena cava and seen on the roof of the left auricle, still unites the two auricles, and represents the original continuity of the two chambers (see Fig. 7, A).

Thus in the right auricle of the human heart there is musculature derived from three sources—(1) from the auricle proper, (2) from the auricu-

lar canal, (3) from the sinus venosus. In the left auricle the musculature arises from (1) auricle proper and (2) auricular canal. All these parts are in the freest muscular continuity.

The Ventricle of the Human Heart.—It is unnecessary in this place to discuss the correspondence of the common ventricle of the lower forms with the divided ventricular chamber of the higher forms. They are developed as outgrowths from the ventricular segment of the primitive cardiac tube; the part which remains undisturbed between the outgrowths forms the interventricular septum. The upper margin of the septum represents the least disturbed part of the lumen of the primitive tube; on it lies the a.-v. bundle.

The Bulbus Cordis of the Human Heart.—This fifth division of the heart is well marked in the primitive forms (Fig. 1, *e*). It is generally supposed to be absent in the mammalian heart, but recently Greil (3) and one of the authors (4) has shown that this is not so; the infundibulum of the right ventricle represents practically the whole of this cavity. The musculature of the bulbus has become replaced entirely or for the greater part by that of the ventricle.

The Musculature of the Sinus Venosus and of the Sino-auricular Junction.—Having thus sketched out, perhaps too briefly, the primary divisions of the heart, we now propose to describe the musculature of the sinus venosus and its connections with the other parts of the heart, more particularly in relationship to two points in physiology. (1) The rhythm of the heart begins in the sinus: does its musculature or any part of its musculature show any peculiar differentiation in connection with this function? (2) What are the muscular connections of the sinus; are they restricted so that a sino-canalar or sino-auricular "block" may occur, as is supposed by Wenckebach, or are they so wide and diffuse that such a block is inconceivable from an anatomical point of view? These two matters we shall discuss in relationship to the human heart, using our comparative material only in so far as it throws light on the questions we discuss.

Taking the latter question first, we may say at once that the musculature of the sinus is freely continuous with that of the auricular canal and of the auricle. An impulse arising in the sinus musculature around the termination of the superior vena cava (Fig. 3, *a*) may spread directly (1) into the musculature of the interauricular septum, and thus to the network in which the a.-v. bundle begins (Fig. 5, *b*); (2) to the vestibule of the left auricle (*m*, Fig. 3), and to the auricle proper along the interauricular bridge (Fig. 5, *c*); (3) to the auricular canal of the right auricle; (4) to the right auricle proper (Fig. 5, *d*). If the impulse commences in the coronary sinus, then it may spread directly (1) to the vestibule of the left auricle; (2) to

the annular fibres of the left auricle; (3) to the annular fibres of the right auricle (Fig. 3, i). Indeed the higher one ascends in the vertebrate scale, the less becomes the amount of the sinus musculature, but the greater the closeness of its connection with the canalar and with the auricular musculature. It therefore appears to us that the sino-auricular "block" cannot be due to an anatomical lesion of a narrow bridge of fibres, but must arise from the depression, probably of vagal origin, of the muscular tissue in this region.
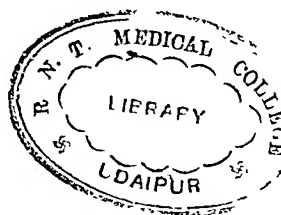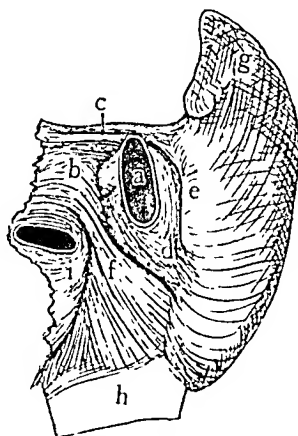
Fig. 5.—Right auricle of human heart viewed from above, to show connections of musculature at the termination of the superior vena cava.

a, superior vena cava cut across; b, vestibule of left auricle; fibres are seen to enter the interauricular septum from the superior vena cava, c, sinus musculature of superior vena cava passing to left auricle (in in fig. 3), d, sinus musculature of superior vena cava crossing sulcus terminalis to right auricle; e, sino-auricular junction; f, muscula ture of interauricular septum submerging sinus, g, appendix, h, inferior vena cava, i, septal fibres passing on to vestibule of left auricle below orifice of pulmonary vein

In Fig. 6 we give two sections of the sino-auricular junction:[1] A is that of the human heart made across the sulcus terminalis in the position shown in Fig. 3, B that of the heart of the turtle. The venous valve (see Fig. 6, B) at the sino-auricular junction is seen to be really a fold of the cardiac tube; the musculature of one side of the valves is derived from the sinus— that of the other is continuous with the auricular musculature. At the free margin of the valve the sino-auricular muscle is continuous (Fig. 6, B, 3). A certain amount of fibrous tissue belonging to the epicardium is enclosed within the folds of the valve; in this an artery is frequently present. By the musculature of the valves an impulse may be freely distributed in the musculature of the auricular canal and of the auricle proper, for at the upper and lower angles at which the valves unite their musculature

[1] We use the term "sino-auricular" in preference to "sino-canalar" because, although a true sino-canalar junction exists on the dorsal side in the most primitive hearts (see Fig. 1), yet in all but these the part of the canal between the sinus and the auricle disappears, and the dorsal junction becomes really a sino-auricular junction. Moreover, as the term "auricle" is usually applied in the mammalian heart to the parts representing both auricular, canal and auricle proper, the term "sino-auricular" is the more appropriate.

spreads out and joins freely with that of the auricular division of the heart (Fig. 6, *B*, *4*). In the mammalian heart a distinct remnant of the sino-auricular junction, so well shown in more primitive hearts, can be recognised (Fig. 6, *A*). In the human heart, as in most mammalian hearts, an artery or arterial circle lies in the junction (Fig. 6, *A*, *2*); the artery is surrounded by fibrous tissue in which are numerous peculiar muscle fibres, some nerve cells and nerve fibres. The nerve cells and fibres we find from dissection to connect with the vagal and sympathetic nerve trunks which form so rich a plexus and exert so powerful an effect at this junction. The

A   **B**
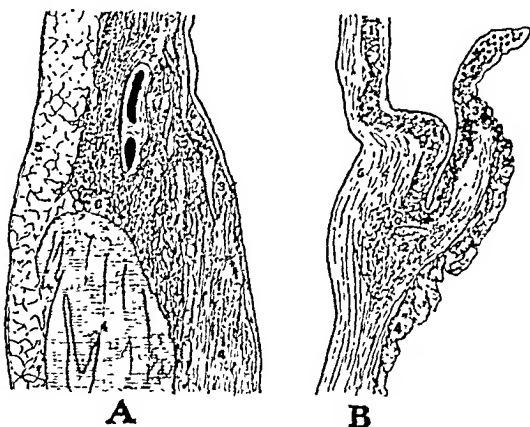
Fig. 6.—A, sino-auricular junction in human heart (position indicated in fig. 3); B, sino-auricular junction in turtle's heart. The figures represent corresponding parts in the two hearts.

A   **B**
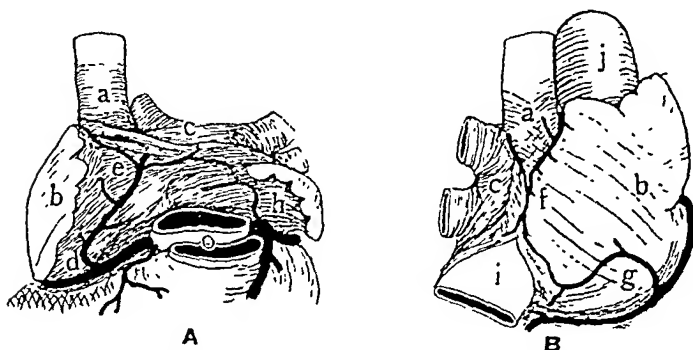
Fig. 7.—Showing blood-supply of the musculature of the sino-auricular junction.

A, aorta and pulmonary arteries removed, exposing auricles from the front; B, right auricle from the side. *a*, superior vena cava; *b*, appendix; *c*, vestibule of left auricle; *d*, artery arising from right coronary and passing to sino-auricular junction; at *e* the artery divides, one branch passing in the junction in front and the other in septum behind superior vena cava; *f*, union of two branches above mentioned in sulcus terminalis; *g*, anastomosing branch from right coronary artery; *h*, anastomosing branch from left coronary artery; *i*, inferior vena cava; *j*, aorta; *o*, aorta and pulmonary artery.

musculature of the superior vena cava becomes continuous with that of the auricle and of the auricular canal both on the outer and inner side of the artery.

Our search for a well-differentiated system of fibres within the sinus, which might serve as a basis for the inception of the cardiac rhythm, has led us to attach importance to this peculiar musculature surrounding the artery at the sino-auricular junction (Fig. 6, A, 2). In the human heart the fibres are striated, fusiform, with well-marked elongated nuclei, plexiform in arrangement, and embedded in densely packed connective tissue—in fact, of closely similar structure to the *Knoten*. The amount of this musculature varies, depending upon how much of the sinus has remained
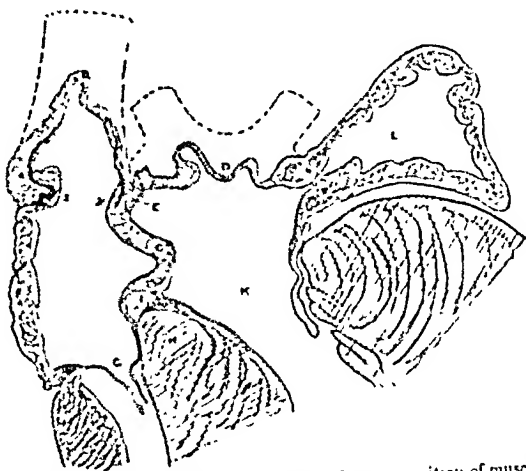
Fig. 8.—Coronal section of the mole's heart, showing position of musculature at sino-auricular junction.

xx, Junction of superior vena cava and right auricle; A, peculiar musculature described in paper at sino-auricular junction; B, section of wall of superior vena cava. the position of the superior vena cava is outlined. C, interauricular septum. D, vestibule of left auricle; the pulmonary veins are outlined. E, similar musculature to A lying at junction of superior vena cava and vestibule. F, wall of right auricle; G, right auriculo-ventricular orifice; H, interventricular septum; I, a.-v. bundle similar in structure to A; K, left auriculo-ventricular orifice; L, left auricle **; canalo-auricular junction

of the primitive type; but in the neighbourhood of the taenia terminalis there is always some of this primitive tissue found. Macroscopically, the fibres resemble those of the a.-v. bundle in being paler than the surrounding musculature, *i.e.* in being of the white variety. They can be dissected out on the superior vena cava in the region corresponding to the right venous valve (a, Fig. 3), and at the coronary sinus in the interval between it and the inferior vena cava and left auricle (b, i, Fig. 3). Another remarkable point in connection with these fibres is the special arterial supply with which they are provided (Fig. 7, A and B). These arterial branches, as noticed by Wenckebach, embrace the sino-auricular junction. It will be seen that they come from both right and left coronary arteries

and form what may be termed the "sino-auricular arterial circle." We might mention also that, in some of the pathological hearts cut by us, sections of this region appeared to show a definite increase in the amount of fibrous tissue present—a fact of considerable importance, since we have found that the fibrous tissue of the *Knoten* and a.-v. bundle is sometimes increased in pathological hearts.

The nature of this remnant is perhaps best exemplified in the heart of the mole (Fig. 8). Here it is seen that at the sino-auricular junction (*A, E*) there is a mass of remarkable tissue. It appears to the eye as a very intimate network of palely stained undifferentiated fibres with a large number of well-stained nuclei. It is totally different from the surrounding musculature, and contains but little fibrous tissue. Although the mass by its connections is undoubtedly muscular, the nerves in the neighbourhood of the superior vena cava appear to come into very intimate connection with it, so much so that we feel justified in stating that a highly differentiated neuromuscular junction occurs at this point. In this heart also the bundle (*I*, Fig. 8) is of absolutely identical structure.

In a section of the heart of the wallaby in this region there is seen under the low power (2″) a mass of fibrous tissue apparently separating superior vena cava from auricle. On closer inspection, however, it is seen that very delicate, palely stained, primitive muscular tissue is enclosed within the fibrous mass.

A section of this junction in the porpoise's heart is interesting. The musculature of the superior vena cava has largely remained primitive in type. The wall of the superior cava consists of alternate layers of fibrous tissue and primitive palely staining fibres. Just at the junction, however, of the superior vena cava and auricle, a network of these fibres and fibrous tissue is formed. In it there is an artery, and two nerve trunks lie close by.

In the dolphin's heart, on the other hand, there is no difference between the greater part of the musculature of the superior vena cava and that of the auricle. But in the region of the taenia terminalis there occurs some loosely-woven fibrous tissue, in the meshes of which are contained an artery and wavy, delicate muscle fibres with well-marked nuclei.

The ram's heart also shows a similar characteristic set of fibres in this region. The tissue in this case shows a marked amount of fibrous tissue loosely interwoven with palely-staining fibres closely resembling those of the sinus of the frog. The remnant also occurs in the hearts of the kitten, rat, and mouse.

From the above it will be seen that the presence of these primitive fibres is remarkably constant. Physiologic experiments have clearly demonstrated that normally the heart's rhythm begins in the neighbourhood of the great veins, and that here nervous influence has a most potent effect (MacWilliam, Engelmann, Hering, and others). The fact, therefore, that there is a constant differentiation of certain fibres in this region, which, moreover,

are in close connection with the nerves affecting the heart's rhythm, leads us to attach great importance to these fibres, and we feel justified in expressing the opinion that it is in them that the dominating rhythm of the heart normally begins.

The Canalo-auricular Junction.—At the ostium of the auricle in the lower hearts the musculature is directly continuous with that of the auricular canal (Fig. 1, 2-2). In them there is a difference in the type of fibre constituting the two parts, those of the auricle being coarser, more striated, and more deeply stained. In the mammalian heart the junction between the parts representing the auricular canal and the auricle proper is also marked by a thickening of the musculature. The fusion of the different systems of muscle fibres, however, is for the most part so intimate that it is difficult to distinguish between them. We could find no trace of any especially differentiated fibres at this junction.

The Canalo-ventricular Junction.—This is the junction of the auricular canal with the ventricle. It has been described by MacWilliam in the heart of the eel. In this heart the auricular ring is connected to the ventricular system by the fibres of the invaginated part of the auricular canal (see 4-1, Fig. 1). This part of the canal shows a differentiation even in the eel. Its fibres differ from those of the rest of the canal in being larger, less striated, staining more palely, and possessing a very large distinct nucleus.

In the frog there is a similar connection all round the auriculo-ventricular orifice below the base of the a.-v. valves, but particularly below the auricular septum. The fibres of the connection are not differentiated from those of the rest of the canal: they are shut off by fibrous tissue from the ventricular system in the upper part of their course, but later on they fuse with the fibres of the innermost part of the ventricular wall. The canalo-ventricular junction in the reptilian heart is similar to that of the fish and of the frog.

In the mammalian heart the auricular ring and the invaginated fibres become profoundly modified. Taking the human heart as a type, we find that the ring can still be traced round the right auriculo-ventricular orifice above the bases of the valves. The fibres are no longer isolated, but can be identified by their structure. No trace of them can be found in the canal of the left auricle. It will perhaps be well to recall the arrangement of the muscular connection between the auricular canal and the ventricle in the human heart. The system begins in the *Knoten*, a small mass of interwoven fibres in the central fibrous body of the heart, having slender connections with (1) the musculature of the interauricular septum; (2) the circular fibres of the right auricular canal. From this arises the main bundle which passes along the upper border of the interventricular septum below the pars membranacea septi. Here it divides into a right and a left division, which pursue a subendocardial course in the right and left ventricles, respectively, and finally fuse with the ventricular muscle. The fibres composing the

main bundle, and more especially its arborisations, vary very much in type in the hearts of different species. In some hearts there is a marked difference from the ordinary ventricular musculature. Such is the case in the hearts of the sheep, ox, calf, cart-horse, pony. In these the main bundle consists of long, delicately striated fibres, with large nuclei. The end arborisations consist of fibres belonging to the Purkinje system. In other hearts the fibres of the bundle and its terminal branches are not so well differentiated from the ventricular fibres. This is the case in the whale, kangaroo, wallaby, dolphin, man, rat, kitten, mouse, shrew-mouse, and pig. In these hearts, however, and especially in the first four mentioned, there is still a differentiation of fibre rendering the bundle quite distinct from the ordinary ventricular musculature. The fibres of the bundle are larger, more delicate, less striated, and stain less deeply than those of the ventricle proper. In certain other hearts, namely, those of birds, the authors have been unable to find any differentiation of fibres in the bundle; the guides to it being its position and its definite demarcation by fibrous tissue. In the bird's hearts examined by us, the a.-v. bundle arises from the auricular ring and dives at once into the interventricular septum.

The a.-v. bundle is the sole muscular connection between the auricular canal and the ventricle; there is no direct connection between auricle proper and ventricle in the mammalian heart. It must be admitted, however, that in one case, namely, in the heart of a rat, the auricular and ventricular fibres appear to come into close apposition in the right lateral auriculo-ventricular region, and undoubtedly represent one of the connections described by Stanley Kent. In the heart of the sparrow also there is a similar close apposition of fibres in this region. This close apposition, however, cannot be looked upon as a connection; the a.-v. bundle is to be regarded as the sole connection between the auricular canal and the ventricle.

The Bulbo-ventricular Junction.—This junction is well marked in the primitive hearts (see 5, Fig. 1). In all a circular groove containing epicardium separates the ventricular from the bulbar musculature, but not completely; the inner or subendocardial layer of ventricular musculature becomes continuous with the bulbar musculature. In the frog's heart this is also the form of connection, but the union is three or four times denser on the dorsal than on the ventral side of the b.-v. junction.

In the mammalian heart the bulbus has become fused with the right ventricle, forming the infundibulum of that cavity. Greil's research on the heart led him to the conclusion that, although the cavity of the bulbus remains, its musculature has been overgrown and replaced by that of the ventricle. The moderator band which passes from the septal wall of the right ventricle to the base of the anterior group of musculi papillares marks the separation of the bulbar part from the rest of the right ventricle. On this band of muscle the right division of the a.-v. bundle descends: that

is, if our identification be correct, the right septal division descends in the position of the bulbo-ventricular junction. There can be no doubt, at least, that there is no bulbo-ventricular separation of fibres in the mammalian heart.

The Morphology of the A.-V. Bundle.—This is the third point which we had in mind during this research. As the result of our examination of the hearts in different branches of the vertebrate kingdom, we have come to the following conclusions in reference to the morphology of this bundle:—(1) The *Knoten* represents the only part of the annular ring of the auricular canal of the primitive heart which has remained undifferentiated in type. The rest of the ring has become differentiated and is imbedded in the other auricular musculature as explained above. (2) The main bundle and its two divisions represent the remnant of the invaginated portion of the auricular canal.

The chief evidence in favour of (1) may be summarised thus —The different position occupied by the *Knoten* in relation to the central fibrous body in the hearts of different animals, *e.g.* of the sheep, horse, and man, points to the fact that in each a different portion of the auricular ring has remained undifferentiated as the *Knoten* The musculature of the *Knoten* resembles in structure the other portion of the primitive canal which has remained undifferentiated, namely, the remnant at the sino-auricular junction, evidenced especially by the hearts of the mole, rat, and ram. Lastly, in the heart of a human embryo (32 mm. long) the auricular ring is clearly seen, and the part which is to persist as the *Knoten* is in close continuity with the ventricular musculature. The ring in this embryo is at the upper part of the interventricular septum, and its fibres are of exactly the same type as persist in the *Knoten* throughout life.

The evidence that the main bundle is the remnant of the invaginated portion of the auricular ring reveals itself as we proceed from the lower to the higher forms. In the eel this part of the auricular canal forms the a.-v. connection, which is all round the auriculo-ventricular orifice. In the amphibian and reptilian heart the connection is still around the whole orifice, but it is thickest at the base of the interauricular septum. In the bird's heart the connection is comparatively large, and is situated solely at the base of the interauricular septum. In the mammalian heart the connection is small, and occupies the upper border of the interventricular septum. It is beyond the purpose of this article to discuss the physiological reason for this restriction of the muscular connection between the auricle and the ventricle to a narrow bundle which measures on the average only 1.5 × 0.8 mm. in diameter; but its persistence in the position which it occupies becomes intelligible when it is called to mind that the upper border of the interventricular system represents the least-disturbed part of the lumen of the embryonic cardiac tube.

Summary

I. (a) The muscular connection in the lower hearts between sinus and auricular canal, and in the higher between the parts of the heart representing them, is intimate. In the latter, fibres pass directly from this junction to the vicinity of the a.-v. bundle.

(b) The canalo-auricular junction is marked by a thickening of the heart wall at this point. The muscular connection is diffuse. In the lower forms there is a difference between the fibres of the two parts, but in higher forms the fusion is so intimate that no difference in the type of fibre can be distinguished.

(c) The canalo-ventricular junction decreases in extent from the lower to the higher forms; in the latter it is represented solely by the a.-v. bundle.

(d) The bulbo-ventricular junction is well marked in the lower hearts. In higher forms the ventricular musculature has replaced that of the bulbus.

II. (a) There is a remarkable remnant of primitive fibres persisting at the sino-auricular junction in all the mammalian hearts examined. These fibres are in close connection with the vagus and sympathetic nerves, and have a special arterial supply; in them the dominating rhythm of the heart is believed to normally arise.

(b) No special differentiation of fibres was found at the canalo-auricular and bulbo-ventricular junctions.

III. (a) The *Knoten* is a part of the primitive auricular ring which has remained undifferentiated.

(b) The main bundle and its branches represent the invaginated portion of the primitive auricular canal.

References

(1) Engelmann, *Pflüger's Archiv*, series of articles, vols. lii., liv., lvi., lix., lxii., lxv. Summary of Literature, vol. lxv., p. 535.

(2) Gaskell, *Journ. of Phys.*, vol. iv, p. 43; *Schafer's Text-book of Physiology*, vol. ii.

(3) Greil, summarized by Hochstetter in *Handbuch der vergleich. u. experim. Entwickelungslehre der Wirbeltiere*, parts 4, 5, 14, and 15. 1903.

(4) Keith, A., *Studies in Pathology*. Aberdeen, 1906.

(5) Keith and Flack, *The Lancet*, August 11, 1906.

(6) MacWilliam, *Journ. of Phys.*, vol. vi., p. 192; vol. ix., p. 173.

(7) Tawara, S., *Das Reizleitungssystem des Saugethierherzens*. Gustav Fischer, Jena, 1906.

(8) Wenckebach, K. F., *Archiv für Anat. u. Phys.* (Phys. Abteil), 1906, p. 297.

Note.—Since the above was written, Dr. J. Mackenzie has kindly drawn our attention to a recently published paper of Hering's, "Über die Automatie des Säugethierherzens," in *Pflüger's Archiv*, Bd. 116, p. 143. It is interesting to note that Hering brings about complete stoppage of the supraventricular parts of the heart by a cut made at the sino-auricular junction in precisely the same position as our section (Fig. 6, *A*).

1908

SIR JAMES MACKENZIE

DESCRIPTION OF AURICULAR FIBRILLATION

SIR JAMES MACKENZIE
Photograph by Emery Walker

(Courtesy Charles C Thomas.)

SIR JAMES MACKENZIE

(1853-1925)

"The Beloved Physician"

—R. McNair Wilson.

*J*AMES MACKENZIE was born at the farm of Pickstonhill, Scone parish, Scotland, on April 12, 1853. His father, Robert Mackenzie, and his mother, Jean Campbell Menzies, had moved there from Perthshire. Near his birthplace stood the ruins of the abbey wherein the kings of Scotland had been crowned since antiquity.

Young Mackenzie was a pupil first at the village school and later at the grammar school at Perth, the latter institution being so old that it was well known in 1153. Mackenzie left school, at his own volition, at the age of fifteen, to become an apprentice in a chemist's shop. He served his apprenticeship here for five years, and it was the inspiration derived by contacts with country physicians who came to the chemist's shop to have their prescriptions compounded that caused Mackenzie to decide to study medicine. After he had served his apprenticeship his employer offered him a partnership in the shop. However, he decided to accept a post in Glasgow as a chemist. He remained for a year and his work at Glasgow confirmed his determination to become a physician.

Mackenzie was twenty-one years old when he went to Edinburgh to begin the study of medicine in the university of that city. He had some difficulty, as he himself said, in passing the examinations of his early years, because his power of memory, on which the examinations depended, was not so good as his power to reason. During this period of his life as well as during his earlier academic training at Perth, Mackenzie exhibited a sense of inferiority. He always felt himself the "dunce" of the school and even later, when he was awarded three medals for superior clinical work at the University of Edinburgh, he still felt that he was a student of the most inferior grade. It is of interest to note how well this sense of inferiority paralleled that of the great American historian, Henry Adams, who twenty years earlier, at Harvard College, felt the same frustration and sense of failure. Adams had a sense of frustration and spiritual weariness all his life, but as he is acknowledged the authority in certain aspects of American history, so Sir James Mackenzie has often been acknowledged as one of the greatest physicians of the twentieth century.

Mackenzie was graduated from medical school in 1878. The year was a conspicuous one in medical history, for it ushered in the opening stages of the battle which Pasteur and Lister were waging in behalf of the germ theory of disease. Mackenzie's application for resident at the Edinburgh Royal Infirmary was accepted and he remained there a year. In 1879 Mackenzie was induced by Dr. John Brown, his former professor in anatomy, to become an assistant in the firm of Dr. William Briggs and Dr. John Brown at the Lancashire town of Burnley in England. At the end of a year's work he was offered a third share in the practice. This he accepted, and the situation was so pleasant for Mackenzie that he remained in Burnley for thirty years.

Early in his practice at Burnley, Mackenzie was summoned to help a young woman in childbirth. She was his own patient. The delivery promised to be easy and uneventful. But during the stages of labor a tragedy occurred: his patient suddenly

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died of heart failure. James Mackenzie, as he turned to break the news of her death to her husband, tasted the bitterest anguish which any doctor can ever experience. He felt responsible for the death of his patient, for there probably had been, long before the time of delivery, some cardiac sign by means of which, had he recognized it, he might have been able to take certain precautions. There and then Mackenzie resolved to specialize in the study of the heart.

He began by studying the mechanism and symptoms which generally are presumed to indicate cardiac conditions in pregnant women. On consulting the literature he found nothing that proved helpful. To find answers to the riddles of the heart, Mackenzie made innumerable studies. In investigating the circulatory conditions before, during, and after pregnancy not only among women who had cardiac disease but also among healthy women, he found changes in the size and position of the heart, murmurs of various types, variations in rate and rhythm, and other departures from what is usually considered the normal. To distinguish types he resolved to ''write'' the pulses of a large number of women and then to study the tracings with the closest possible attention. At that time there was available the Dudgeon sphygmograph or pulse writer. With the use of this instrument Mackenzie soon confirmed his contention that several different varieties of irregularity occurred in the tracings. But what did these different waves in the tracings signify? Which were truly dangerous ones, and which were innocuous? There did not seem to be an answer.

Another fact confused him. He found out that the pulsations of the vessels of the neck were just as varied as were the waves of the pulse. Mackenzie felt that if tracings of the pulsating vessels of the neck could be obtained, some factors might be correlated that would aid in the solution of the problems which beset him. He therefore modified the Dudgeon apparatus and made the first crude tracings of the pulsations of the vessels of the neck.

Mackenzie's work on the tracings was interrupted by a most pleasant occasion. His marriage to Miss Frances Bellamy Jackson took place on September 13, 1887. The honeymoon was spent in Italy. The marriage itself was a happy one, Lady Mackenzie later declaring that ''No woman ever had such a generous love and companionship given her.''

As has been suggested, Mackenzie's difficulty in employing the mechanical registration of pulsations was to interpret the various waves he was able to inscribe. At first he attempted to determine the answers to these enigmas by questioning the leading physiologists of his time, but they could not answer his queries. Some time later Mackenzie devised a new, portable recording instrument. Using the radial pulse as a standard and a pill-box for the drum he was able to construct an apparatus which he carried around in his pocket. About this time the ink polygraph[1] and the electrocardiograph made their appearance. However Mackenzie for several years preferred to use his miniature instrument because he believed it was simpler and better adapted for general use. Later, when he found out that the signs he had discovered with his various instruments were also detectable by the finger of the physician, he used the instruments less frequently than before. Mackenzie resented the attitude of the medical profession in too completely accepting the ink polygraph and later the electrocardiograph. He believed that physicians were more interested in the exactness of mechanical devices than in the ends for which these mechanical devices were constructed.

Earlier, Mackenzie had discovered a method of determining how two of the heart's four chambers were acting at any given moment. He had also made the discovery

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[1]Later, Mackenzie, with the help of a Mr. Shaw, a watchmaker of Padiham, perfected the ink polygraph which is today known the world over as Mackenzie's Ink Polygraph.

that pregnant women tended to have irregularities of the pulse. Suddenly the idea occurred to him to determine what the various chambers of the heart were doing when that organ was beating irregularly. When his next patient presented himself with a marked irregularity of the heart he was able to prove that in some cases of irregular action, the ventricle contracted sooner than normal. The auricle, in maintaining its normal rate and rhythm, actually beat after the ventricle. Later, Mackenzie demonstrated that this form of irregularity, the extrasystole, was in itself of no serious consequence to the patient. Although identification of the extrasystole was in effect a negative discovery, it was most important. It was proof that many patients presenting themselves with this type of cardiac irregularity need not be alarmed and that they could lead normal lives.

In about 1890 Mackenzie's attention was directed to another group of peculiar irregularities. A woman whom he had first seen in 1880, when she was suffering from an attack of rheumatic fever, again presented herself for treatment. He discovered that her heart had been damaged, and that the damage was the result of the attack of rheumatic fever in 1880 and subsequent attacks in 1883 and 1884. A presystolic murmur was audible. Narrowing of the mitral valve continued. Mackenzie maintained careful observation of this patient, noticing that in 1898, with alarming rapidity, his patient grew worse. An amazing thing occurred at that time. The presystolic murmur vanished! After considerable thought, Mackenzie came to the conclusion that the auricles of the heart had ceased to beat, and that it had been the beat of the auricles forcing the blood through the narrowed passage in the heart that had caused the presystolic murmur. A year later the patient died, and on examining the heart Mackenzie verified his belief. He found the auricles to be enormously distended and greatly weakened. He gave to this type of irregularity the provisional name "paralysis of the auricle." It is now known as "auricular fibrillation," because of Mackenzie's research and the subsequent researches of a favorite pupil, Sir Thomas Lewis.

Mackenzie noted these conditions among a number of patients and his recognition of the disappearance of auricular contractions instituted a landmark in the history of medicine. It was the central feature of much subsequent work on the elucidation and grouping of the various anomalies of the rhythm of the heart. It further prepared the way for Mackenzie to establish, together with Cushny, the conditions necessary for efficient digitalis therapy. The results of Mackenzie's early observations appeared in his "Study of the Pulse" (1902).

The unique experiences of Mackenzie in cardiology have been admirably set forth in his monumental work, "Diseases of the Heart," of which the first edition was published in 1908. His careful study of semeiology appeared in 1909. Another important work, "The Future of Medicine," appeared in 1919. This volume is mainly autobiographical and expresses the wisdom which made him famous. Besides revising his small and large books on the heart, Mackenzie in 1923 contributed a superb monograph on the disease of which he was destined to die, "Angina Pectoris." Another important work, "The Basis of Vital Activity," was completed shortly before his death in 1925. It was not published, however, until 1926.

After practicing medicine at Burnley for nearly thirty years, Mackenzie went to London in 1907, at the age of fifty-four. In London, although he earned only about $600 during his first year there, he soon became an outstanding Harley Street consultant. He continued his studies and somehow found time to write several books which we have mentioned. Within a short space he was elected a member of the Royal College of Physicians. He was further honored by a fellowship in the Royal Society, and in 1915 he was knighted. In 1918 he retired from active practice.

The next year he established at St. Andrews in Scotland the Institute for Clinical Research.[2] Mackenzie defined the object of the Institute's work as "the prevention of the diseases that are common among the people." The reports of the Institute have been regarded as most valuable contributions in the systematic study of symptoms and disease.

Shortly after coming to St. Andrews, he was stricken by angina pectoris. Although he knew of his condition, he continued to work unceasingly at the Institute. He revised his great work on "Diseases of the Heart," lectured in London, Edinburgh, Dundee, and Aberdeen. He wrote several articles for medical journals and continued working on his several books.

His final months were severely painful, and he suffered numerous cardiac attacks. He died on January 26, 1925, foretelling the period of his death with uncanny certainty. Some weeks before he died, he directed Dr. John Parkinson to examine his heart after his death. After Mackenzie's death his brother, Sir William Mackenzie (now Lord Amulree), corroborated this request. The examination was performed by Dr. John Parkinson, assisted by Dr. J. W. Linnell and Dr. David Waterston. The latter wrote an account of the observations, which we reprint.

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[2]Now known as the James Mackenzie Institute of Clinical Research.

CHAPTER XXX

AURICULAR FIBRILLATION*

The importance of recognizing auricular fibrillation.—The most important of the continuous abnormal rhythms is that which is due to fibrillation of the auricles. The recognition of this condition and the symptoms associated with its presence, is the most important discovery yet made in the domain of the functional pathology of the heart; and few physicians are aware of its significance. The symptoms directly due to auricular fibrillation, and the symptoms of heart failure induced by this condition, are so clear and definite, that we have little difficulty in recognizing this condition as a distinct clinical entity. Its recognition is not of mere academical importance, but is of the greatest practical value; for when we recognize the various symptoms, they afford us grounds for a sure diagnosis, a safe prognosis, and for a rational line of therapy in a large proportion of cases of serious heart failure. The great frequency of its occurrence renders it imperative that all practitioners should become familiar with its symptomatology; for 60 or 70 per cent of all cases of serious heart failure met with in practice owe the failure directly to this condition, or have the failure aggravated by its presence. Some of the symptoms have been overlooked in the past, while the significance of others has not been appreciated. Moreover, the response of hearts affected with auricular fibrillation to remedies differs so much from the response of all other forms of heart action, normal and abnormal, that the recognition of its characteristics materially alters the views universally held as to the action of drugs upon the heart.

Type of case showing auricular fibrillation.—Before setting out in detail the features characteristic of auricular fibrillation, it might be convenient to appreciate the kind of case which shows this condition. The most common evidence is an irregular action of the heart of a very disorderly kind. It is that form of irregularity so frequently met with in the elderly, and in patients with hearts damaged by previous rheumatic infection. In the latter class, the association of irregular hearts with mitral stenosis has long been recognized; and, on account of this association, the irregular pulse is sometimes described as the "mitral pulse," with which all clinicians are familiar.

While senile and rheumatic hearts are those most frequently affected by this condition, there are numerous cases in which auricular fibrilla-

*Sir James Mackenzie first described this condition in 1908. In the first edition of his book, *Diseases of the Heart.* We have chosen to reprint from the third edition of this work, published in 1914, pp 211-236.—F. A. W. and T. E. K.

auricular fibrillation. As the auricle was found distended and thin-walled at the post-mortem examination, I came to the conclusion that the disappearance of the signs of auricular systole was due to the auricle having become distended, atrophied, and paralysed. This view I put forward in a book on the pulse, which I published in 1902. Shortly after this was published, I had a series of cases, some of which I had watched for years, and at the post-mortem examinations the auricles were not thinned, but were hypertrophied. With this fact before me, I saw that
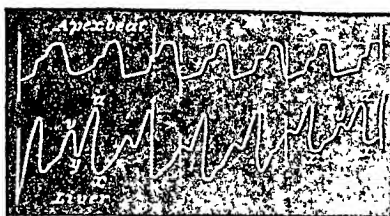
FIG. 119.   The liver pulse shows a well-marked wave (a) due to the auricle (Case 48. 1892).
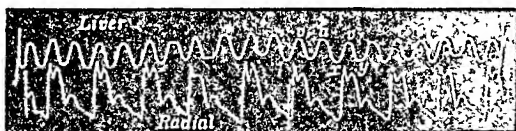
FIG. 120.   There is still a well-marked wave in the auricle (Case 48. 1897).
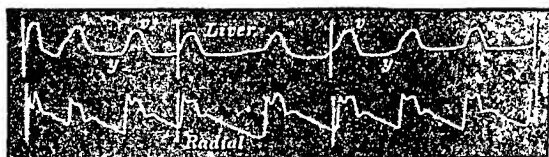
FIG. 121.   Showing the irregular rhythm, characteristic of auricular fibrillation. When compared with Fig. 119 and 120, it will be seen that there is no auricular wave in the liver pulse, and the heart's action is irregular (Case 48. 1898).

my previous explanation could not be correct; for the fact that the auricles were hypertrophied, indicated that they must have contracted during the years that I had watched them, and when there had been an absence of all signs of auricular activity. As it was clear that the auricles could not have contracted during the normal period—that is to say, immediately before ventricular systole—the only alternative I could see was that they contracted during ventricular systole. As, in the meantime, I had studied several hundreds of cases and had seen this condition start under a variety of circumstances, particularly in individuals with frequent extrasystoles, I put forward the view that ventricles and

auricles contracted together, and assumed that the stimulus for contraction arose in some place that affected auricles and ventricles simultaneously. As at this time I could not conceive of any other possibility to explain the facts, I suggested that the stimulus for contraction arose in the auriculo-ventricular node; and I called the condition "nodal rhythm," under which name the clinical aspects of auricular fibrillation are described in the two editions of this book, the first being published in 1908.

With the advent of the electrocardiograph, we obtained a more accurate method of recognizing the contraction of the chambers of the heart. When electrocardiograms were taken of the cases that I had called nodal rhythm, my clinical observations were verified, inasmuch as no evidence of the normal auricular systole was found. In cases where the heart periodically became disorderly in its rhythm, and where I was able to demonstrate that the auricular form of venous pulse was present with the regular heart action, and the ventricular form during the period of irregular action, the electrocardiograms also showed evidence of auricular contraction during the normal period of the heart's action, and a disappearance of the normal auricular activity during the period of irregular action, fully confirming the observations I had made on nodal rhythm.

The attention of other observers had also been arrested by some of the clinical features of this condition. Thus Hering, in 1903, separated from among other irregularities the irregularity peculiar to auricular fibrillation, and called it the *pulsus irregularis perpetuus*. He was mainly concerned with the physiological aspect of the subject, and did not recognize the full clinical picture, with the disappearance of all signs of auricular activity. Many other observers had noted the "positive" venous pulse, and in attributing it merely to tricuspid incompetence they had failed to appreciate its real meaning, and so missed the significance of its appearance.

Although the disappearance of the auricular contraction was the feature that puzzled me in these cases, I realized that my explanation of it, as being due to synchronous contraction of auricles and ventricles, was far from being established; and I endeavored to interest others in the subject, who might investigate the matter by experimental methods, and find out, if possible, what the auricle was doing. Cushny was the first to suggest that auricular fibrillation might be a factor of clinical importance; and in 1906 he and Edmunds drew attention to the resemblance of the radial tracings in a case of paroxysmal irregularity in the human subject to the tracings from a dog, in which they produced experimental fibrillation of the auricles. On reading this communication, I was struck with the idea; and on a visit Professor Cushny paid to me in Burnley in 1906, he discussed with me the probability of auricular fibrillation being the cause of the irregular

heart action in certain cases of "nodal rhythm," and he agreed that certain small waves, which I had recognized in the jugular pulse of one case (Fig. 122), were due to the fibrillation of the auricle.

I published, in 1907, tracings with this explanation, but I failed to appreciate the real significance of what auricular fibrillation was; I thought it only a passing event; and I practically gave up the idea that it was at the bottom of these cases that went on for years. Lewis had been pursuing an inquiry clinically and by experiment into the nature of cardiac irregularities, and had produced experimental fibrillation in the dog. In 1909 he took graphic records of the venous and arterial pulses. With the onset of

Fig. 122. A tracing from a patient with auricular fibrillation, showing small fibrillary waves (*f*) in the jugular tracing (Case 44).
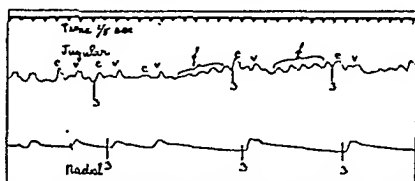
Fig. 123. The jugular tracing shows coarser fibrillary waves (*f*).
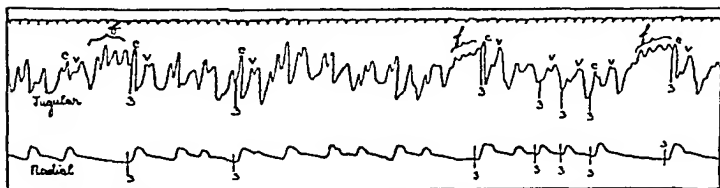
Fig. 124. The jugular tracing shows fibrillary waves (*f*) of different sizes.

fibrillation, he found that the arterial pulse became irregular, and the venous pulse changed from the auricular to the ventricular form. Pursuing his investigations further, Lewis was able to detect in the electrocardiogram of experimentally produced fibrillation, certain oscillations during ventricular diastole, which were induced by the fibrillating auricle. Examining more critically the electrocardiograms of typical cases of nodal rhythm which I sent to him, he found these oscillations also present, and demonstrated their correspondence with the small fibrillation waves I had noted in the jugular pulse.

When Lewis placed these facts before me, I had no hesitation in abandoning my views, and accepting the fact that these cases owed their abnormal action to auricular fibrillation; and I now recognize that the reason those evidences of auricular activity, to which I have referred, disappear, is because the auricle ceases to act as a contracting chamber.

Rothberger and Winterberg had independently, in 1909, drawn attention to the fact that in *pulsus irregularis perpetuus* the electrocardiogram corresponded to that of auricular fibrillation experimentally produced.

In the investigation which I have been carrying on for so many years, I was not content merely to discover the mechanism by which the irregularities were produced, but I always kept before me the bearing these symptoms had on the patient's present and future state, and what indications they gave for treatment. To this end, I made careful notes of all attendant circumstances, such as the patient's history, the size of the heart, the degree of heart failure, the response to treatment, and the future progress of the case. The result is, that though I failed to recognize the nature of the altered rhythm of the heart, yet from my notes of a great number of individual patients, many of whom I had watched for a number of years, I had been able to study many of the characteristics of this group and to recognize the clinical features.

Up till 1908, these observations were carried on in my work as a general practitioner. Since 1909 I have reviewed the whole subject, first at the Mount Vernon Hospital, and then at the London Hospital, with the assistance of my colleagues, and we have verified the main features I had previously recognized, and extended our observations. Although we can recognize many salient features of the condition, there is still a great deal of work to be done, before a full knowledge of the change in the heart's action under this new rhythm can be acquired.

What is auricular fibrillation?—The term "fibrillation" is applied to a curious condition of the muscle fibres of the heart, where the individual fibres, in place of contracting in an orderly and simultaneous manner during systole, contract rapidly and independently of one another. The auricle, when in a state of fibrillation, presents an entirely different aspect from what it does during its normal action.

"The walls of the auricle stand in the diastolic position; systole, either complete or partial, is never accomplished; the wall, as a whole, is stationary, but careful examination of the muscle reveals an extremely active condition; it appears to be alive with movement; rapid, minute, and constant twitchings or undulatory movements are observed in a multitude of small areas upon its surface" (Lewis).

When the ventricle passes into fibrillation, the circulation is at once brought to a standstill; and MacWilliam has suggested that this is probably the cause of sudden death in the human subject. When the auricles

pass into fibrillation, death does not ensue, for the fibrillation cannot pass along the bundle which connects auricle with ventricle.

Conditions inducing auricular fibrillation.—In experiment, auricular fibrillation can be produced by electrical stimulation of the auricular wall. In the human heart it is found to arise under a variety of conditions. It is probable that it is produced by altered nutrition of the muscle. Thus, I detected it in 1892 in a patient recovering from a mild attack of rheumatic fever, and there was no other evidence that the heart was affected. The attack passed off after some hours, and the youth has grown up into healthy manhood with no evident lesion of the heart. I have seen it appear in the heart in pneumonia, during the attack and during convalescence with disastrous results in both cases. Digitalis can induce it in predisposed cases. I have known it to occur intermittently in a fatal case of infective endocarditis, and Price has shown its occurrence in a fatal case of diphtheria, and G. A. Sutherland in a severe attack of rheumatic fever. Post-mortem examination in these cases showed that marked changes of an inflammatory nature had occurred in the walls of the auricle.

Effort, sometimes slight, and sometimes violent, may provoke auricular fibrillation. This occurs most frequently in the middle-aged or elderly, or in those with some old rheumatic affection Thus, a healthy and vigorous member of our profession at the age of 50 years ran rapidly for 200 yards, and was seized with an attack which lasted for two hours. This was ten years ago, and he is still well and actively engaged in his work. In many cases, these attacks lasting for a short period are apt to recur with increasing frequency until they become permanent. While they are occurring intermittently, they are often easily provoked by effort, though they may not infrequently arise from no apparent cause. Thus, in one man under my care at the Mount Vernon Hospital, the heart would be detected beating irregularly several times a day, the irregular period lasting from half an hour to two hours. This irregularity was due to auricular fibrillation, as shown by records by the polygraph and electrocardiograph. He himself was not conscious of the altered rhythm, nor was there any recognizable cause for the onset.

We are not yet in a position to decide, with sufficient accuracy, the nature of the changes in the heart-wall which favour the occurrence of auricular fibrillation. In the hearts which I have had examined, which showed auricular fibrillation during life, there has been found in the auricle and ventricle, an increase of fibrous tissue and of nucleated cells in the muscular walls (see Cases 48, 49, and 51). In most cases, there is probably some definite change which predisposes to this condition, and it only needs an adequate stimulus to provoke it. This stimulus may be of a varied kind, for while the onset can frequently be traced to violent bodily effort, it often occurs when there is no excessive effort. At pres-

ent, we can only say that one predisposing condition is certain organic changes in the muscle wall of the auricle.

Duration of auricular fibrillation.—In the majority of cases when auricular fibrillation sets in, it persists for the remainder of the individual's life. I have watched individual cases for over thirteen years, in whom it was constantly present (see Case 43). In many cases, it may appear for a few hours, and may never recur, or it may recur at infrequent intervals for some weeks and months, and then disappear. Many cases of paroxysmal tachycardia owe the paroxysms to auricular fibrillation, and in such cases it may last for a few seconds, a day or two, a few weeks, or even months (see Case 51). As a rule, however, when it is intermittent in its appearance, the tendency to its recurrence becomes greater, till finally it becomes permanently established.

Effect on the ventricles.—In fibrillation of the auricle, the stimulus for contraction arises no longer in the sino-auricular node, but in the fibrillating fibres of the auricle, and is transmitted to the auriculo-ventricular node in an irregular manner. It is probable that the manner in which the ventricle is affected depends on the power of the node and bundle to receive and transmit the auricular stimulations; for I found that in some of my cases the ventricular rate varied very much, sometimes being rapid and sometimes slow (see Cases 44, 51, and 54). It might be suggested that with the onset of auricular fibrillation the ventricle takes on a rhythm of its own, and indeed at one stage of my investigations I had suggested an idio-ventricular rhythm. But, in experiment, if after fibrillation has been set up, the bundle is cut, the ventricular rate at once alters, and the ventricle assumes its own peculiar slow rhythm.

Rate.—Changes in the rate and rhythm of the ventricle, and in the size of the heart, are very common with the onset of auricular fibrillation. In several cases, I have detected these changes shortly after its inception. I have found the greatest difference in rate, ranging from 40 to 130 beats per minute. It is but seldom that we get the opportunity of seeing auricular fibrillation start, as the patient is frequently unconscious of the change in the heart's rhythm, though some recognize the curious sensation of fluttering. The patient may consult us because of the distress which may sooner or later appear, and then we usually find the rate remarkably increased, generally between 110 and 140 beats per minute and over. I have met with a number of cases, in which the rate has become slower on the inception of auricular fibrillation. When fibrillation has arisen as the result of the administration of digitalis, the rate has been infrequent (Case 92). If digitalis slows the heart in fibrillation through its action on the vagus and the auriculo-ventricular bundle, we might assume that the slowing occurs in consequence of some affection of the auriculo-ventricular bundle. In support of this view I have observed

a case for many years, in which there was persistent increase in the interval between the auricular and ventricular systoles, and in which at one time there was partial heart-block. When the patient's auricle started to fibrillate, the ventricular rate fell from 60 to 40 beats per minute, and has continued at this rate for nine years. Cases 80 and 81 show a slow pulse-rate, but in these there was present complete heart-block. In Case 79, the heart fell suddenly to 40 beats per minute and was quite regular; the slow rate persisted for a fortnight, when it suddenly increased in rate and the auricle resumed its normal action. In this case, during its normal action, there were no evidences of heart-block.

Rhythm.—When the auricle passes into fibrillation, the ventricle usually becomes irregular in its action. The alteration is sudden, as shown not only by experiment but by clinical observations, in cases in which I have actually observed the change in the heart's action. The cessation of fibrillation can be recognized by the return to a regular rhythm; occasionally the return is accompanied by a few irregular beats, due to extra-systoles. Though many speak of this condition as the *pulsus irregularis perpetuus,* I have seen a number of cases where the rhythm of the ventricle was regular. In the majority of such cases, the rate was under fifty beats per minute. In several instances, the slow regular action has been induced by digitalis. In a case recently under observation, the rate under digitalis fell from 110 to 70 beats per minute. Prior to the administration of digitalis, the rhythm was very irregular, but when the rate fell to 70 beats per minute, it was quite regular. The patient had no jugular pulse, but by the electrocardiograph, Lewis demonstrated that auricular fibrillation was present.

The character of the irregularity as seen in the pulse is a completely disorderly one, in the sense that the interval between the beats is ever varying, two successive beats being seldom of the same length. Although, as a rule, there is a distinct relation between the size of the beats and the length of the preceding pause, the longer pauses being followed by bigger beats, not infrequently this is not so, big beats sometimes following very short pauses.

Many other conditions produce continuous irregularities, so that we have to be careful not to form our opinion on the irregularity by itself.

The size of the heart.—In the great majority of cases, a considerable enlargement of the heart follows the inception of auricular fibrillation. Though the auricles are often greatly distended, it is not possible to tell from clinical examination, how much of this enlargement is due to the auricles, and how much to the ventricles.

With the onset of fibrillation, the increase in size does not take place at once, though, in a few cases of periodic fibrillation, I have seen the heart increase greatly in size within a few hours of the onset, and the enlargement would disappear within a few hours after the cessation of

the fibrillation. As a rule, little change in the size of the heart takes place at first, and if the heart is capable of maintaining an efficient circulation little increase in size may be detected for years. In the majority there is an inability to do the work efficiently, so that gradually the heart's strength becomes exhausted, and an increase in its size follows. With appropriate treatment, a considerable diminution may take place, but this is by no means constant. I have been surprised at its persistence in cases of old-standing auricular fibrillation, in whom a very striking improvement of the heart's condition has taken place as the result of treatment. In most cases we have carefully studied at the Mount Vernon Hospital, and at the London Hospital, we have failed to detect any decrease in the size of the heart, in patients who had suffered from extreme failure, and who had made a surprising recovery.

The jugular pulse in auricular fibrillation.—The size of the jugular pulse is extremely variable, in different cases, and in some individuals at different times. This is in a great measure due to the amount of distension of the right side of the heart and of the great veins, and also to the rate of the heart. In some slow-acting hearts, great waves can be seen extending up the neck with each contraction of the ventricle, and there is no difficulty in recognizing their nature. In other hearts beating at more rapid rates, these waves are also evident; one cannot be always sure of their nature from inspection alone, but a tracing will show them to be of ventricular form. When the veins are less full, and the rate frequent, it is utterly impossible to differentiate the waves that may appear in the veins. Even in a graphic record some difficulty may be met with, but if the time of the waves be accurately placed in the cardiac cycle in the manner already described, as a rule the features of the tracing can be recognized. In many cases, the character of the ventricular waves shows a curious difference. Thus some slow beats will show a high wave at the same time as the carotid pulse (marked c in the tracings), and a great fall during the mid-systole of the ventricle, with a wave towards the end of systole, which ends as usual with the opening of the tricuspid valves. With the more rapid beats the fall during mid-systole disappears, the characteristic wave of the ventricular venous pulse is shown. The fall is due to the dragging down of the auriculo-ventricular septum during ventricular systole.

Fibrillation waves.—There is an additional feature, which is also of value in recognizing the presence of auricular fibrillation, and that is the presence of the small waves caused in some way by the fibrillating auricle. They are not present in every case, nor always perceptible in those cases that show them. When present, they are most evident during those long pauses of the ventricle which are so frequent in auricular fibrillation. These fibrillation waves are of a variable size, sometimes very minute,

as in Fig. 122, and sometimes very coarse, as in Fig. 123. In Fig. 124, they will be found to vary in size and duration at different times.

The electrocardiogram in auricular fibrillation.—While the normal electrocardiogram shows features similar to those in Fig. 16, a great number of differences can be obtained, each due to some abnormal action of the heart. So far as the electrocardiogram of auricular fibrillation is concerned, in addition to the irregular action of the ventricles, the records show some very characteristic signs, the chief being a total disappearance of the peak, P, due to the auricle. The variations R and T, due to the ventricle, maintain their characteristic form. Between the ventricular beats, the records may show a series of small movements, which we now recognize as being due to the fibrillating muscle of the auricular wall. This absence of the auricular movements and the presence of the movements during ventricular diastole are the means, by which auricular fibrillation can be recognized in the electrocardiographic record. Further, there is also the characteristic disorderly rhythm.

Dr. Lewis tells me, that the small movements shown in the electrocardiogram are not constant, but in every given case they come and go for no apparent reason. In this they differ from the movements in auricular flutter, which are constant. There appears to be some relation between these movements and the fibrillation waves found in the jugular tracing, and as these come and go, in an unaccountable way, it is probable that they are both due to the same factor.

Changes in the heart's murmurs.—I have already dealt with the evidences of auricular activity, obtained by graphic and electrocardiographic methods. The evidence of auricular activity obtained in the clinical examination, apart from the graphic records of the jugular pulse, is limited to the murmurs of mitral stenosis. We must bear in mind that stenosis of the mitral valve is a gradual process, at first not recognizable until a certain degree of narrowing has arisen. This narrowing obstructs the flow of blood from auricle to ventricle, and gives rise to a murmur on the contraction of the auricle. A presystolic murmur is evidence of a contracting auricle, and is usually an indication not only of an obstruction to the flow of blood from auricle to ventricle, but also of gradually progressing fibrotic changes in the valves and around the mitral orifice. With progressive narrowing, the presystolic murmur becomes louder and longer, while a new murmur may appear after the second sound. This diastolic murmur is due to the obstruction of the flow of blood through the mitral orifice at the end of ventricular systole. It is faint and short at first, but with further narrowing of the mitral orifice it increases, till it fills up a great portion of the diastolic period of the cardiac revolution, and may run up to the presystolic murmur. When this happens, the diastolic period is filled up entirely by murmurs. We may take it that the appearance of this diastolic mitral murmur is al-

ways an evidence, that the progressive narrowing of the mitral orifice has reached an advanced stage. But the fibrotic changes that cause mitral stenosis are not limited to the valvular orifices, but are also present in the muscular walls of the heart. These changes in the auricular wall predispose to auricular fibrillation, and this may arise at different stages. With the onset of fibrillation, a change takes place in the character of the murmurs.

If a presystolic murmur, due to the auricular systole, was present prior to the onset of the fibrillation, it at once disappears when the pulse becomes irregular. If a diastolic murmur, due to mitral stenosis, has been present, it persists, because it is caused, not by the systole of the auricle, but by the inrush of blood from the auricle into the ventricle when the ventricle relaxes after its systole. I wish to emphasize this change in the character of the murmurs in mitral stenosis with auricular fibrillation, for even those who detect the clinical symptoms of auricular fibrillation do not seem to have grasped the significance of the change in the murmurs. I have carefully studied a large number of cases of mitral stenosis with fibrillation, and many of them have come to an autopsy, and in none of them have I detected a presystolic murmur of the crescendo type. Where a murmur has preceded the first sound, it has filled the whole diastole when the rate was rapid. When the heart's rate is rapid in auricular fibrillation, this diastolic murmur often fills up the whole space between the second and first sounds, and it may simulate, and is often taken for, a presystolic murmur due to the systole of the auricle. If, however, this murmur be noticed during one of the long pauses, which occur frequently in most cases, or when the heart's rate becomes slow, it will be found that this murmur follows the second sound, but pauses some little distance before the first sound, and there is a silence immediately before the first sound in the place where the crescendo presystolic murmur due to the auricle should appear.

This is brought out in the diagram in Fig. 125, where A and B represent the sounds and murmurs in mitral stenosis before and after auricular fibrillation. In B it will be seen that there is no presystolic crescendo murmur, and that the diastolic murmur fills up the whole of the space between the second and first heart sounds, when the interval is short (x,x); but when the interval is long (y,y) the diastolic murmur does not reach the first sound, and there is a silence before it.

From these considerations, we can, in the great majority of cases, conclude that auricular fibrillation is present when there is a diastolic mitral murmur without a presystolic murmur. As a rule, the irregular action of the heart is also suggestive, but in many cases of mitral stenosis with auricular fibrillation, when the patient is under digitalis, the heart becomes slow and almost, or even quite, regular.

The explanation given here occurred to me in 1897, as an outcome of the study of the features of Case 48. Since that time I have continued the observations and verified it repeatedly; but I have found the greatest difficulty in convincing physicians of the clinical facts. The murmur filling up the interval between the second and first sound is invariably looked upon as presystolic, while the long diastolic murmur present when the heart is slow (see Fig. 192*), is not infrequently taken to be aortic; on sundry occasions I have heard physicians express surprise that at the post-mortem examination there has been no aortic lesion, but mitral stenosis in cases with the long diastolic murmur. Quite recently Dr. Lewis has taken records, by means of the electro-phonograph, of cases of auricular fibrillation, and his results have confirmed the explanation given above.

Auricular fibrillation and digitalis.—Not the least in importance of the discoveries resulting from the recognition of auricular fibrillation as a clinical entity, is the light that is thrown upon the action of drugs of
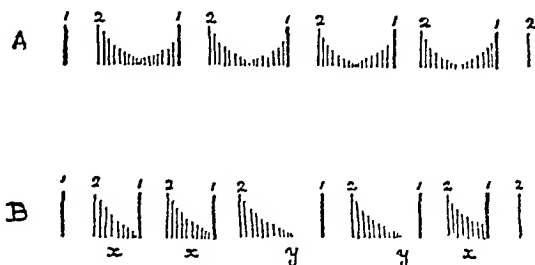
Fig. 125.—Diagram illustrating the change in the murmurs in mitral stenosis when auricular fibrillation occurs. The perpendicular lines 1 and 2 represent the first and second sounds of the heart, and the shading between the second and first sounds represents the diastolic and presystolic murmurs with a regular rhythm (*A*). In B auricular fibrillation causes the rhythm to be irregular, and when the diastolic period is short as at *x, x, x*, the diastolic murmur fills up the whole interval. When, however, the diastolic period is lengthened as at *y, y*, the diastolic murmur does not fill up the whole period, and there is then a silence before the first sound when the presystolic murmur was heard before fibrillation set in.

the digitalis group. I can only here refer, briefly, to a few points which I have been able to elucidate. I think every one who has carefully studied the description usually given of the effects of digitalis on the human heart, cannot but be struck with the absence of agreement among the different writers, as to the manner of its action, its dosage, and the best preparation. In some instances, some peculiar reaction which the observer may have noted is looked upon as the characteristic effect of digitalis but the mechanism of this peculiar reaction has not been understood. It is a good many years since I was struck with the varied reactions, which I obtained from the use of digitalis. I collected a great number of cases; in some I got a definite reaction on the heart, while in others no reaction was obtained. When I separated these into groups,

*Appears in chapter beyond that reprinted in this book.—F. A. W., 1940.

[781]

I saw that the probable reasons for the varied reactions in the human heart were, that digitalis gives a reaction according to the nature of the lesions from which the heart is suffering. It will be observed that if this is found to be correct, we can at once understand how the physiologist and experimental pharmacologist have missed the most important effects of digitalis, for, so far as the heart in experimental work is concerned, they cannot reproduce the conditions under which the physician has to employ the drug.
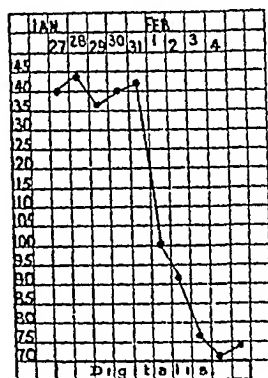
FIG. 126. Chart showing a typical reaction of digitalis in a case of auricular fibrillation with severe heart failure. The administration of the tincture of digitalis began on January 27, in doses of 15 minims four times daily.
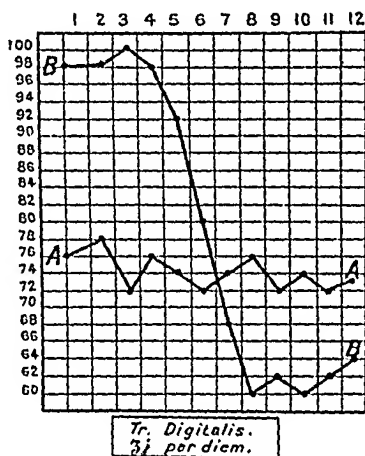
FIG. 127. Chart showing the effects of similar doses of tincture of digitalis on six cases of mitral stenosis with the normal rhythm (A) and six cases of mitral stenosis with auricular fibrillation (B). The figures at the side represent heart-beats; those at the top, days. In each case the digitalis was continued till the heart became slowed, or until nausea or vomiting occurred. The average quantity before an effect was produced was 7 drachms (1 drachm per diem). This had little or no effect on the heart-rate in the patients with the normal rhythm (A), while there was a rapid decrease in the rate in the patients with auricular fibrillation (B).

It is not only in auricular fibrillation that digitalis acts beneficially, for there are many other conditions which benefit by it; but cases of fibrillation stand apart from all others in regard to their response to this drug. All cases of auricular fibrillation are not responsive; for there are factors which render certain hearts unsusceptible, as the presence of fever or extensive fibrous degeneration. It is in certain cases, where there is a fair amount of healthy muscle that its almost specific action is seen. It is some ten years, since I realized this peculiar response to digitalis. When I was appointed to the Mount Vernon Hospital and London Hospital, I seized the opportunity to start a series of observations under conditions, which permitted a degree of accuracy unattainable in

private practice. In these observations, the same drug and the same dose were given to patients with and without auricular fibrillation. With only rare exceptions, all the cases that showed a marked effect upon the heart were cases of auricular fibrillation; for, although the other cases might exhibit some benefit from the use of the drug, they never showed the same tendency to slowing of the heart's rate.

The slowing effects of digitalis are shown in a very striking manner in those cases of auricular fibrillation, where heart failure set in with a great increase of rate of the heart. The chart (Fig. 126) is a good illustration of these types. The patient from whom the chart was obtained, suffered from extreme heart failure, and the rate of the heart was 140 per minute and very irregular. She was given one drachm of digitalis per day, and after five days the pulse-rate fell in the manner shown in the chart. At the same time, there was a remarkable improvement in the patient's general condition.

The difference in the reaction of hearts affected with auricular fibrillation and those with the normal rhythm is well brought out by the chart in Fig. 127, where the average heart-rate from six cases of mitral stenosis with auricular fibrillation is compared with six cases of mitral stenosis with the normal rhythm. The record begins with the rate on the day previous to administration of the drug; and it will be seen that the rate in the cases with auricular fibrillation is greater than the rate in cases with the normal rhythm. This, I may remark in passing, is a point of some interest; for cases of heart failure with mitral stenosis with the normal rhythm, rarely, have as rapid a pulse as those with mitral stenosis with auricular fibrillation.

In each case the tincture of digitalis was given, one drachm per day, and was continued till nausea or vomiting ensued.

Digitalis in some hearts induces auricular fibrillation. When this occurs, the rate of the ventricle becomes greatly decreased (see Case 92).

Effect on the heart's efficiency.—A great many patients in whom auricular fibrillation had occurred, suffered from lesions of the heart, which impaired its efficiency. In all these, the occurrence of fibrillation at once increased the impairment, and the symptoms of heart failure became intensified. In others, where there had only been a slight impairment of the cardiac efficiency, the onset of auricular fibrillation speedily provoked symptoms of extreme heart failure, while in others little difference could be detected. In a few cases, the onset of auricular fibrillation has only slightly embarrassed the heart in its work.

To a great extent the symptoms of heart faliure arise most markedly in those in whom the change in the auricular action has affected the ventricle, particularly in increasing its rate. In a few cases, marked limitation of the heart's powers of response to effort has followed, with relatively slow acting hearts.

[783]

The symptoms of heart failure commonly produced are of the same kind as arise in heart failure from other causes, for example, shortness of breath on exertion and consciousness of the heart's action, particularly when some effort is made. With increase in the failure, oedema of the legs and lungs sets in. The face becomes livid, the patient cannot lie flat in bed, but has to be propped up. The liver becomes enlarged, while the veins in the neck may become engorged, and the pulsation in them becomes extremely marked. With these changes, dilatation of the heart and great increase in the rate may be detected. As a rule, the onset of these symptoms is slow and gradual, but occasionally they may set in with great rapidity; they quickly disappear if the heart reverts to its normal rhythm.

Cause of heart failure in auricular fibrillation.—However it may be brought about, the onset of fibrillation embarrasses the ventricle in its work, and in all probability the degree of heart insufficiency that results depends upon the extent of concomitant damage of the ventricles, and the amount of embarrassment caused by any valve lesion that may be present. There can be little doubt that the orderly action of the auricle in regulating the supply of blood to the ventricle, and in stimulating it in a normal manner, results in a more efficient action of the ventricle than the variable and irregular stimulation to contraction. When the ventricle is rapidly and irregularly stimulated to contraction, there results a gradual exhaustion of the strength of the ventricle, and evidences of heart failure supervene. In cases that have drifted and died, where I have had a post-mortem examination, there has been found extensive fibrous degeneration of the heart muscle (Cases 49 and 51). On the other hand, I have seen so many individuals with auricular fibrillation who have led strenuous lives, engaged in hard manual labour, that I infer that in them the muscle of the ventricle had not been seriously damaged (Case 44).

Clinical characteristics. *The patient's history.*—The conditions which induce auricular fibrillation vary; but there are two classes among whom it is very frequent—namely, patients with a heart affection following upon rheumatic fever (frequently associated with mitral stenosis), and elderly patients with fibrous degenerative changes, usually spoken of as "senile." It may be found in the young and middle-aged, with no history of infection. It is customary in rheumatic hearts to take into account only the valvular changes, while as a matter of fact the really serious element is the slow, insidious change in the muscle that has started during the attack of rheumatic fever, and which finally provokes fibrillation of the auricle.

The patient's sensations.—Many people become conscious of the heart's action when it departs from its normal rhythm. Thus, extra-systoles are sometimes recognized by the individual's consciousness of the long pause,

or of the big beat which follows the pause. The patients liable to paroxysmal tachycardia, are conscious of the attack by the feeling of a gentle fluttering sensation in the chest. When the attacks of tachycardia are due to auricular fibrillation, this fluttering is also present; but usually it is not a continuous fluttering sensation, but is interrupted by thumping sensations, due to the occasional occurrence of bigger beats. This consciousness of the heart's action is frequent in cases of auricular fibrillation, the patients being conscious of the fluttering and irregular action of the heart. In many cases, where the heart does its work efficiently, these sensations are not perceived, unless the heart is submitted to over-exertion, or when it begins to fail.

Character of the pulse.—The symptom by which the clinical observer can most readily recognize this condition, is by the character of the pulse, the rhythm usually being irregular, and the irregularity of a very disorderly kind. Irregularities, apart from those due to auricular fibrillation, usually have a distinctive character, as the irregularity in the heart of the young, where variations in rate coincide with phases of respiration, as the intermittent pulse, or the irregular heart due to extra-systoles, the irregularity breaking in on an otherwise regular rhythm, unless it occurs alternating with a normal pulse-beat. In auricular fibrillation, as a rule, the pauses between the beats are continuously changing and two succeeding beats are rarely of the same strength, or the pauses between the beats of the same duration. The character of the irregularities will be better recognized from the radial tracings, as shown in Figs. 122, 123, and 124.

Symptoms of heart failure.—The sign which usually calls attention to auricular fibrillation is the patient's consciousness of his limitation. The signs of this limitation, however, are not peculiar to or characteristic of auricular fibrillation, but are common to heart failure induced by other conditions. These signs of heart failure may range from a slight breathlessness on exertion, to dyspnoea of the most severe kind, accompanied by dropsy, enlarged liver, and the symptoms associated with extreme heart failure.

As a rule, the onset of symptoms of heart failure is slow and gradual, due in a measure to the individual's persisting in living in his usual manner, though the heart is hampered by the abnormal action. On the other hand, the onset of heart failure may be very rapid. Within a few hours of the occurrence of auricular fibrillation, the distress of the patient may be very severe, the countenance dusky, the heart dilated, orthopnoea, and a feeling of distress is experienced. I have seen these phenomena arise rapidly in cases, where the fibrillation occurred intermittently, and the relief experienced by the patient, when the heart resumed the normal rhythm, was as remarkable and striking as the onset of the suffering. At once the patient knows that the heart's action has altered, and he breathes easier, and the feeling of distress disappears.

Within a few hours, the heart and liver have become reduced in size, the lividity of the face has gone, and the tenderness of the chest-wall and over the region of the liver speedily disappears.

In many cases, the persistence of heart failure is accompanied by wasting, the patient sometimes losing a good deal of weight in a few months. Accompanying this, there is usually a certain amount of flushing of the cheeks, usually of a dusky colour, and occasionally a slight sallow tinge of jaundice. These symptoms with an enlarged liver may be mistaken for sarcoma of the liver.

Auricular fibrillation and angina pectoris.—Amongst the more common signs of heart failure, there is one which I have but rarely seen—namely, definite attacks of angina pectoris. As I have already stated, pain and hyperalgesia are not uncommon in the heart failure associated with auricular fibrillation, while I have met with typical attacks of angina pectoris in only a few cases. In quite a number of cases where the patient had angina pectoris, the attacks ceased with the onset of the fibrillation. Needless to say, the onset of auricular fibrillation in those cases induced such embarrassment to the heart's action, that the patients all drifted, and only lived a few months after its onset. The onset of fibrillation may be associated with angina pectoris (Case 50).

Prognosis.—We must bear in mind that auricular fibrillation is in reality a symptom of some myocardial change, and that, to be logical, we should only consider it from the point of view of a myocardial affection. We are at present so ignorant of myocardial disease, that we are forced to put one symptom forward as if it were in itself a disease. Illogical as this seems, it has its use, for the occurrence of auricular fibrillation induces such a profound change in the heart's action, affecting its efficiency, reacting on the ventricle, and modifying its behaviour to drugs, that we are compelled to look upon it as a condition apart. Considering the variety of conditions that induce auricular fibrillation, it is difficult to state briefly its prognostic significance. In referring to the pathological lesions associated with it, I showed that they were of a very diverse kind in nature and in degree. It is in all probability the extent of these pathological changes which determines the prognosis of auricular fibrillation, and an attempt should be made to estimate their extent. If we look upon the inception of the new rhythm as in itself embarrassing the heart in its work, then the maintenance of an efficient circulation depends on whether the heart is able to do its work, when hampered by the new rhythm. That this is the fundamental question will be recognized, when we study the effect of fibrillation in certain individuals. I have repeatedly seen individuals in whom fibrillation occurred for a short time, and in whom heart failure set in with an extraordinary rapidity, the patient becoming breathless, having to sit up in bed, the face becoming livid, the heart dilated, and the liver swelling, within a few hours

after the onset of fibrillation. With the restoration of the normal rhythm, these symptoms quickly disappeared. When fibrillation became permanent in such individuals, the signs of heart failure persisted sometimes in spite of all treatment, till death supervened in a few weeks or a few months (Cases 51, 52, and 53).

On the other hand, I have repeatedly seen fibrillation set in, and the individual be altogether unconscious of its presence. These cases may go on for years with little inconvenience; but the majority after some years gradually show signs of a limitation of the field of cardiac response, and their future depends upon how they respond to treatment, and on their ability to diminish the amount of their bodily work, and to live within the limits of the heart's strength (Cases 43, 44 and 45).

Much more frequently there is a considerable limitation of the heart's power of response to effort; and if the usual life of the individual be pursued, without appropriate treatment, there is a great tendency for the heart gradually to fail. There is no doubt that the onset of fibrillation can lead directly to a fatal termination, or, rather, can be associated with conditions that lead to death. Thus, one of my patients died suddenly a few days after the inception of auricular fibrillation. Another one fell down dead six months after its inception. I have seen a number of other patients die suddenly, who had auricular fibrillation, but some of these suffered from a considerable degree of heart failure. It has appeared to me probable that in these cases the ventricle has passed into fibrillation, as MacWilliam suggested. This view is probable also from the fact, that the histological changes in the ventricle were similar to those in the auricle in some of the cases of sudden death.

The usual mode of death in auricular fibrillation is a steady advance of the heart failure, as shown by the breathlessness on exertion, orthopnoea, dropsy, and enlargement of the liver, etc., sometimes with an absolute failure of response to all forms of treatment. Thus, I have seen death ensue in this manner a few weeks after the inception of fibrillation (Case 52), and others have drifted on for a few months (Cases 50 and 51), while some have led a somewhat chequered career for a number of years, seldom fit for much bodily exercise.

In giving a prognosis in cases of auricular fibrillation, it is necessary to appreciate a good many other things, besides the mere presence of the fibrillation. It is necessary to form an opinion of the extent of the changes that have led up to the fibrillation, and in many cases to find out how long these changes have been going on; as, for instance, the date of an attack of rheumatic fever; and how the patient comported himself before the onset of fibrillation; if, for instance, he was liable to attacks of heart failure, in which case such attacks point to a tendency to exhaustion which may be aggravated by the fibrillation. Amongst valvular lesions, cases with affections of the aortic valve are usually

seriously embarrassed, particularly in aortic regurgitation, when, prior to the onset of fibrillation, there had been evidences of failure. The character of the murmurs present in mitral stenosis will shed light upon the progress of the disease as already described. When auricular fibrillation sets in, it is necessary to observe the accompanying changes in the heart, and the way in which it maintains the circulation. Thus, an increase in the size of the heart, or a rate over 120 beats per minute, usually leads to a speedy exhaustion of the heart's strength. I have occasionally met with an individual with a heart-rate of 100 and 120 per minute, with no increase in the size of the heart, who suffered little inconvenience; but as a rule any rate over 90 beats per minute tends to induce dilatation and consequent exhaustion. On the other hand, when there is little increase in rate, or even when the rate is somewhat slower than normal and the response to effort good, the prognosis is usually very favourable.

In those with symptoms of manifest heart failure, prognosis depends to a great extent on the way in which they respond to treatment. I have already described the action of drugs of the digitalis group in patients with fibrillation, and I will show later, more fully, the action of other drugs in these cases, and the response of the heart to them, and what an important bearing it has upon prognosis.

I have already said that there are a great many individuals with fibrillation who lead useful and energetic lives, and whose capacity for work is little, if at all, impaired by the new rhythm. In such the prognosis is distinctly good.

There are, however, so many exceptions to these details, that a clearer insight may be gained, by looking at each case from a broader standpoint, of how the heart responds to effort, not, however, ignoring the details, but giving them their due consideration. It is in estimating this reserve power that we get the most valuable information. It may be taken for granted that if distress is induced by exertion, so long as the exertion is persisted in, it will ultimately lead to serious heart failure. On the other hand, when individuals with fibrillation are able to undertake the work equal to that done by a perfectly healthy man, there is proof of such a degree of healthy heart muscle and freedom from valvular or muscular embarrassment, that a good prognosis can be given.

We meet, however, with such varying degrees of exhaustion, that an estimate must be acquired of the amount, though it is impossible to describe with accuracy what that amount may be. Even with distinct limitation of the heart's power, as shown by the response to effort, the prognosis may still be favourable, so long as the patient lives within the limits of his powers, avoiding such efforts as cause him distress or exhaustion.

In transient attacks of auricular fibrillation, the attacks usually tend to become more frequent, until auricular fibrillation becomes permanently established. The prognosis of such cases depends on the way the circulation is maintained, this to be estimated in the manner already described. Transient attacks may appear for a short period and then disappear entirely. In two of my cases, I detected a transient attack of auricular fibrillation twenty years ago, and the patients still lead vigorous and active lives. From the recognition of such cases, we can conclude that auricular fibrillation is not of necessity a sign of extreme damage.

A most valuable aid in prognosis may be found in observing how the patient responds to treatment. In sudden attacks of severe heart failure, when the heart's rate is over 120 per minute, it will be well to suspend judgment, until the reaction to digitalis is found out. Many such cases respond speedily to digitalis, and with the resultant decrease in the heart's rate a remarkable degree of recovery may ensue, so that the patients may be able to undertake laborious work, so long as the rate is kept down by the digitalis. This would seem to imply that the exhaustion is mainly brought about by the ventricle being stimulated to too great an activity, and that the slowing enables the ventricle to get more rest, and so regain a measure of strength. From this result, we can also gather that the ventricular muscle must be fairly healthy, and we can estimate, within certain limits, the amount of healthy muscle by the degree of recovery.

Treatment. *General.*—When any individual with heart failure presents himself for treatment, it may be taken for granted that the individual has been undergoing a greater amount of exertion than the heart has been capable of performing without undue exhaustion. Hence the exhaustion of the heart's strength has been brought about in the first place by overwork. It may be that the amount of work has been small, as measured by what a healthy heart can perform, but when a heart is hampered by an inherent defect, such as auricular fibrillation, and the organic changes in the valves and muscle so commonly associated with this condition, the heart may be capable of a very limited amount of effort. With this conception of the cause of heart failure, the first and obvious course to pursue is to ease the heart of its work. In doing this much discrimination is required, and a thorough inquiry into the patient's mode of life has to be made in order to find out what circumstances, such as overwork, sleeplessness, digestive trouble, pain or work, may have provoked or aggravated the heart failure. These have to be attended to in every case, and relief may be at once afforded with the removal of the disturbing or exhausting cause.

The use of digitalis.—While it is important to attend to such circumstances in heart failure with auricular fibrillation, as in all other forms, there are circumstances in cases of heart failure with fibrillation which when appreciated help greatly, not only in the restoration of the heart's

strength, but in the prevention of heart failure. I have already dealt with the reaction of hearts affected with auricular fibrillation to digitalis from a physiological standpoint; it is in treatment of auricular fibrillation that we find the great value of this drug, and I cannot speak too highly of its therapeutic action.

It is seldom that I have been able to say that I have saved a patient from immediate peril by the use of drugs; but this I can say with confidence, that I have repeatedly seen patients in evident peril of death removed rapidly from danger, and restored to a condition of comparative health, and fit for work by the judicious use of digitalis. The manner of its application needs, however, very careful attention, for it is a drug that needs to be applied on certain definite lines, if full benefit is to be obtained from its action. I think it necessary to insist upon this point, for the somewhat "rule-of-thumb" methods of its use, so generally employed, fail to get the full amount of benefit which this drug is capable of bestowing.

To understand the action of digitalis, it is necessary to appreciate the manner in which heart failure progresses in cases of auricular fibrillation, and the way it is controlled by digitalis. It may be taken for granted that when a patient with auricular fibrillation has a pulse-rate, or, to be more accurate, a ventricular rate, of 90 beats per minute and over, he will in course of time gradually lose strength, his heart will become more feeble, and the evidences of heart failure will become more severe. This process may be very gradual, but it is very sure. On the other hand, heart failure may set in rapidly, more especially when the heart's rate rises to 120 and 140 and over. The severity of the failure, however it is brought about, compels the patient to seek rest, and we generally find such patients in bed, sitting up and breathing in a laboured fashion, with considerable distress, the heart usually dilated and the face of a bluish tinge, and possibly with dropsy and pulsation of the liver. In all such cases the prompt administration of digitalis is urgently called for, and, if given in sufficient doses, relief may be obtained in a few days, the relief being accompanied by a remarkable slowing of the pulse-rate. When this is accomplished, or when there are other signs of a sufficiency, the digitalis should be stopped for a few days, and resumed in small doses when the rate begins to increase. The rate of the pulse should be watched, and the quantity sought for which keeps the heart about 70 beats per minute. It is seldom advisable to keep the rate under 50 beats per minute, although in some cases the patient feels fittest when it is at a rate of about 50 beats per minute. In this, we must be guided by the patient's sensations, and the manner in which he responds to effort.

Even when patients suffer from only a moderate degree of heart failure, and are able to go about, it is well to place them under the influence of this drug if the pulse-rate is over 90 beats per minute, and in some

cases if it is over 80 per minute. My usual procedure in such cases is to attend to any circumstance that may aggravate the heart failure, and then to give the patient digitalis until the pulse-rate is reduced. If the failure is of some severity, I put him to bed until the proper effect is obtained, but where it is less in degree I permit him to go about his affairs.

In all cases where the heart has been sufficiently reduced in rate, I find out the quantity of the drug that is necessary to keep the heart at the rate, at which it can perform its work with the greatest efficiency. In doing this, the patient's sensations are of the greatest help, whether he is confined to bed or attending to his affairs. He readily appreciates the change in his response to effort, and some such symptom as a disagreeable action of the heart or breathlessness, can be employed as an indication that the heart's strength is being exhausted. Once the patient understands the meaning of these sensations, he is generally quick to perceive what digitalis does for him, and its administration can usually be left quite safely in his hands. On such lines, I have seen many people lead useful lives for long periods of years with no bad effects, except when they have not taken the drug in sufficient quantities to keep the heart at the required rate.

The foregoing line of treatment is applicable chiefly to cases in whom auricular fibrillation has arisen recently, or where the heart failure is of recent date. In more advanced cases, when the condition has induced from time to time periods of heart failure, and there has appeared the change that accompanies chronic heart disease, such as persistent shortness of breath, enlarged liver, and dropsy more or less continuous, the persistent use of digitalis may still tend to restore a measure of strength to the heart and give relief, enabling the individual to lead a useful life, though at a lower level, for an indefinite period.

In the search for an appropriate line of treatment in old-standing cases, I have used many methods and many drugs, often with little or no benefit, but in a certain proportion of apparently hopeless cases I have seen extraordinarily good results following the use of digitalis, pushed until a reaction was obtained, and then stopped for a time, and again resumed, time after time. Not infrequently, after it has seemed useless to continue the drug, I have seen the individual acquire such an amount of strength as would scarcely have been anticipated.

As the conditions preceding auricular fibrillation and producing it are all due to changes in the heart muscle of a slowly progressive nature, it is easy to recognize that the heart's strength cannot always be restored, and that as the amount of efficient muscle becomes reduced, a period is reached when no method of treatment is of avail.

Method of administration.—A great diversity of opinion is to be found in regard to the form in which digitalis should be given, and also in regard to the dosage. So far as I have worked out the subject in regard

to auricular fibrillation, I see no reason for giving the preference to any particular preparation. The best and most assured way, in cases of marked failure, is steadily to push the drug, whichever form be employed, until a reaction is observed. Usually the digestive system is the first affected, loss of appetite, nausea, vomiting, or diarrhoea being set up, the patient usually feeling ill and miserable. If the digitalis is effective on the heart, as a rule a marked slowing of the pulse is found at the same time, or even before any digestive disturbances arise. In some cases, a slowing of the pulse is the first sign of a sufficiency. When this stage is reached, I always stop the administration of the drug for a few days. In a day or two, patients feel remarkably well and bright, and if nausea was present, it disappears. The heart-rate is carefully observed, and when the rate shows signs of increasing, half-doses of the drug should be given, and the dose increased or diminished according to the manner in which it affects the rate, the object in view being to give just the amount which enables the heart to carry on its work with the greatest efficiency. As I have previously stated, the patient himself by his own sensations speedily acquires the knowledge of how much of the drug is needed, and by attending to his own experiences, he will soon find out the smallest dose which is needed to give the best results.

A good deal of my work has been done with the tincture of digitalis; and I may say that I have used this preparation for over thirty years, and have never yet come across an ineffective preparation, my standard being the reaction in susceptible individuals. Professor Cushny has tested experimentally a number of samples from the Mount Vernon and London Hospitals, and has found each sample effective.

The quantity I usually start with, where the failure is marked, is one drachm of the tincture per day, in doses of fifteen to twenty minims. This is steadily pushed until a reaction is obtained; then it is stopped and employed in the manner already described. Usually a reaction is obtained within a week, sometimes in a few days. Where there is great distress and more urgency, I give as much as two drachms of the tincture daily, and then get a reaction in two or three days.

I have frequently used Nativelle's digitalin granules, and find them also very efficacious. I have found that one of these granules is equal to fifteen minims of the tincture.

Other drugs, such as strophanthus and squills, have the same effect as the digitalis, and in some cases they may cause less digestive disturbance; but in the majority of cases I have found that when the digitalis is ineffective, so also are these drugs. In many cases, the effects of digitalis are less disagreeable than these other drugs.

In some urgent cases, it may be necessary to produce a reaction more speedily, though I have rarely failed to get a reaction in good time by

digitalis by the mouth. In order to obtain a speedy reaction, strophanthin or strophanthone may be injected into the veins. In a series of observations which have been carried out at the Mount Vernon Hospital and at the London Hospital, it has been found that in auricular fibrillation with a pulse-rate of over 140 per minute, intravenous injections of strophanthin ($\frac{1}{250}$ gr.) can reduce the rate and give relief in five or eight hours, but I am of opinion that it is only in very exceptional and urgent cases that this method is required.

Danger in the administration of digitalis.—For a long while, I was at a loss to understand the warning of authorities as to the danger of sudden death from administration of digitalis. Of recent years I have obtained an inkling into the cause of sudden death. I have been shown tracings of the slow pulse with characteristic coupled beats that occur under digitalis with auricular fibrillation, and have been informed that the patient died suddenly. On inquiry, it was found that, notwithstanding the evidences of a sufficiency, the drug had been continued in large doses. I was once asked to see a man who was said to be dying from heart failure. He had to sit up in bed and breathed heavily; his face was livid. He had dropsy, an enlarged liver, and a large and irregular heart beating at the rate of 130 to 140 per minute (auricular fibrillation). I told his doctor to push the digitalis till he showed evidences of a sufficiency, either by the slowing of the heart or nausea, and then to stop it. After five days he telephoned me that the patient was wonderfully free from distress, could lie flat, was a good colour, and the dropsy had almost gone, the pulse-rate being between 70 and 80. I told him to stop the digitalis for a few days, and if the pulse then increased to give smaller doses, and find out the exact quantity which kept the rate about 80. Three days later, he telephoned to say that the patient had been going on well, but that morning, during the doctor's visit, the patient fell back and died. I asked the doctor if he had stopped the digitalis and he replied in the negative, saying that, as it had done him such a lot of good, he had continued it, in spite of my directions.

On making inquiries in a few other cases where I had heard of sudden death, I had no difficulty in recognizing that they were cases of auricular fibrillation, in which digitalis had been pushed after it had affected the heart. Seeing that I have been following this line of treatment by pushing the drug till I get evidences of its action, then stopping it and resuming it later, for over fifteen years, and have never had a sudden death, I am disposed to think that just as we recognize the danger of pushing chloroform beyond a certain stage, so there is danger when digitalis is pushed too far, whereas if the indications I have given are followed, such a catastrophe as death need not occur.
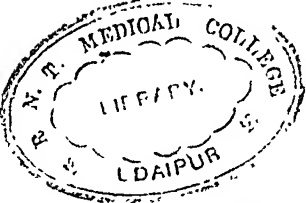
SIR JAMES MACKENZIE'S HEART[*]

By

DAVID WATERSTON

With an Account of His Clinical History by JAMES ORR
And Notes on the Pathological Histology by D. F. CAPPELL

From the James Mackenzie Institute, St. Andrews, and the Department of Anatomy, University of St. Andrews

The following description of Sir James Mackenzie's heart has been prepared in accordance with his desire, expressed to myself and to other friends, that after his death his heart should be examined to ascertain what information it furnished upon the symptoms that he had experienced. He died in London on January 25,[†] 1925, aged 72. Some weeks before his death he told Dr. John Parkinson that he wished him to make a post-mortem examination. This request was confirmed, after his death, by his brother, Sir William Mackenzie, now Lord Amulree. The examination was performed some fourteen hours after his death, by Dr. John Parkinson, assisted by Dr. J. W. Linnell. The heart was removed and subsequently sent to me at St. Andrews for further examination. Dr. Parkinson noted that nothing abnormal was found in the pericardium.

In order to correlate the clinical symptoms with the pathological condition, it has been necessary to compile an account of Sir James Mackenzie's illness. This has not been easy, for, like so many other doctors, he had not been under the care and observation of a medical man from the commencement of his illness. His own case is referred to both in his book on angina pectoris (Case No. 28) and in that on diseases of the heart, as well as in the Reports of the St. Andrews Institute.

While he was in London, from 1908 to 1918, he mentioned to Sir Thomas Lewis that his anginal history began suddenly. While he was in St. Andrews, from 1918 to 1924, engaged in founding the Institute for Clinical Investigation which bears his name, he was on many occasions examined by Dr. James Orr, and discussed with him his condition. Dr. Orr also saw him during several of the attacks of angina, from which he suffered with increasing severity as the years went on. After his re-

turn to London in 1924 he was not under the care of any medical man until very shortly before his death, when he was seen by Dr. Parkinson and Dr. C. M. Anderson.

Dr. Orr, who had seen him during the whole of his stay in St. Andrews, has written the account of the clinical history which follows. My colleague, Professor D. F. Cappell, undertook the histological examination of the blood vessels and heart muscle, and his notes on them are included in the description.

CLINICAL HISTORY AT ST. ANDREWS

The medical life history of Sir James Mackenzie is the story of the onset and gradual progress of angina pectoris from sclerosis of the coronary arteries. He had a mild attack of typhoid fever in 1880 and an occasional attack of renal colic in his later years, but suffered from no other illness.

With the exception of a tendency to extrasystoles commencing at the age of forty, the first evidence of real cardiac involvement was in 1901, at the age of forty-seven. This was a heart attack with irregularity of the pulse, which occurred after running 300-400 yards. In his own description of this attack (Mackenzie, 1925) he notes that he "was conscious of a slight fluttering sensation, but suffered no distress of any kind." The pulse rate during this attack, which lasted two hours, was 90 per minute. A tracing, taken by himself, showed auricular fibrillation. During the next four or five years several attacks of this kind occurred, mostly after a full meal or when walking up a hill; they lasted from ten minutes to half an hour and never caused any distress or limitation in his powers of walking.

The earliest symptom of limitation of effort was noticed by himself in 1907, and was represented by a slight feeling of constriction, hardly amounting to pain, in the upper part of the chest on severe continued exertion, and which soon ceased with rest. There were long periods when it was not experienced at all. For two years he was conscious of slight pain on effort under certain conditions, such as walking after a full meal or on a cold day. This pain he described as preceded by a sense of tightness or constriction, such as used to pull him up when running a race in boyhood.

In 1908, at the age of 55, Mackenzie experienced his first severe attack of cardiac pain. It occurred at night when resting, and followed a period of dining out at frequent intervals. The pain was across the chest and down the left arm; it lasted two hours and varied in severity. Mackenzie further notes that in this attack "he could not be still but had to move about." After 10 grains of veronal sleep was obtained, and next day he was quite well and free from pain, though walking in the cold or

after a meal still produced discomfort of an anginal type. This gradually became more noticeable, and by 1911 there was definite limitation of effort, though pain could be avoided by careful regulation of effort. From this time until the end of his life a somewhat anomalous symptom was present, to which he often referred, namely, that while a sustained effort produced pain, a sudden effort produced breathlessness without pain.

Mackenzie came to St. Andrews in 1918, and at that time was able to walk at any pace from his home to the Cottage Hospital without discomfort, a distance of two miles. In 1919, when I first examined him, he was still able to do this and could play a round of golf regularly. The heart was then ¼ inch external to the mid-clavicular line, the sounds were closed and well spaced, and except for an occasional extrasystole, the rhythm was regular. Blood pressure was 156/92 mm. At this time he was also affected with intermittent claudication on continuous walking. He had first noticed this ten years previously after a rapid four-mile walk, but in 1919 it was evident after a short half-mile walk. The posterior tibial pulse was well felt on both sides. During the next five years this symptom was much less pronounced owing to the fact that pain in the chest occurred in response to a smaller effort than was necessary to produce claudication.

In 1922 limitation of effort prevented his playing golf, and even walking became difficult though by careful regulation of effort, severe pain was, in the main, avoided. A few very severe attacks occurred, like that in 1908, while resting. The most severe of all, in 1923, happened while he was sitting in his study in the afternoon, and lasted nearly an hour; it was little influenced by nitroglycerine and was followed by extreme exhaustion. In August 1924 Mackenzie returned to London, and by this time only the gentlest of exercise was possible. Death followed a very severe and prolonged anginal attack in January 1925. As has been already mentioned, Dr. John Parkinson saw him shortly before his death, and has supplied the following note:

"On January 24 and 25, 1925, he suffered severe and prolonged attacks of anginal pain, and Dr. C. M. Anderson was called during the night. At 4:30 A.M. on January 25 he had morphine subcutaneously, gr. ½, and chloroform inhalation for about an hour. It was necessary to repeat both at 8:20 P.M. on that day. At this time the pulse was 100 and regular and there was Cheyne-Stokes breathing. I did not myself see him until 10:30 P.M. that night (January 25), and he was then asleep. About 1 A.M. on January 26 he awoke free from pain and perfectly conscious and composed. He conversed cheerfully with Lady Mackenzie and me for a few minutes and then said he felt sleepy and soon he slept. At 4 A.M. his breathing changed and became irregular with long pauses, and a few minutes later the pulse stopped. There were no indications of pain at the end."

The heart was uniformly enlarged. Its weight was 18 ounces (510 g.).

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The left ventricle was a large and thick walled chamber. The muscular wall, for the most part thick and firm, was 27 mm. in thickness near the base. At the apex, as usual, it was thin, and only 3 mm. in diameter. In colour it was somewhat pale. In its substance were several small whitish patches of fibrous tissue, in size from a pin's head upwards. In the anterior wall, 30 mm. above the apex, there was a patch of fibrous tissue 8 by 3 mm. and another patch of similar structure and size lay in the substance of the posterior wall, about midway between apex and base. At the apex there was a small recent haemorrhagic infarction involving the deeper part of the muscle wall, covered by a nodular reddish brown mass of clot the size of a cherry stone which projected into the cavity of the ventricle. Section through this and the adjacent wall showed that the nodular tissue extended into the substance of the muscular wall, which was here reduced to a narrow margin 3 mm. thick.

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The aorta had been divided 6 to 7 cm. from its root. At the point of division the lumen was cylindrical, measuring 33-34 mm. The ascending aorta showed a bulging to the right side (the bulb of the aorta) by which the diameter was increased to some 45 mm. The interior of the ascending aorta showed extensive yellow mottling in patches, some separate and some discrete, 3-4 mm. in diameter. Near the root the mottling formed an arborescent pattern. The surface of these mottled areas was slightly raised. In thickness the wall measured 3.5-4 mm., but in places it measured 6 mm. On the posterior wall of the interior of the aorta was a large yellowish raised patch beginning about 35 mm. from the root of the aorta and extending beyond the level at which the vessel had been cut. In this area there was very considerable thickening of subintimal tissue and the tunica intima readily separated off from the other coats. The root of the aorta showed comparatively little pathological change. There were small thin yellow patches of atheroma round the root of the right coronary artery and adjacent to the orifice of the left coronary artery, but the lumen of these vessels was not materially narrowed. Except for the large area mentioned, the wall of the aorta was pliable and showed no general pathological alteration, there being only slight subintimal atheroma.

Section of the patches on the wall showed atheromatous changes of the intimal and subintimal coats. External to the smooth endothelium of the intima was a firm, pale yellow layer some 3 mm. in thickness; external to this the darker coloured and almost unchanged tunica media. There was little, if any, calcification in the subintimal thickenings.

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Coronary arteries.—Both of the arteries and their branches were the seat of advanced and widespread degenerative changes, which had caused thickening of the wall of, especially, the medium-sized and smaller vessels, and diminution of their lumen. The arteries most affected were those in the anterior ventricular furrow, of which there were two, one from each coronary stem. These vessels were so thickened and calcified that their lumen was almost obliterated. A recently occluded vessel was not found as a cause for the infarction mentioned above.

Right coronary artery.—Near its root the external diameter of this vessel was 9 mm. Its wall was greatly thickened and the lumen, oval in outline, measured 2 by 3.6 mm. The thickening involved mainly the sub-intimal coat and also the tunica media, and the wall was firm and rigid. Section of the wall showed patches of degenerated cheesy material in the centre of the thickened areas, and the changes involved almost the whole circumference of the vessel.

From near the root of the artery a branch, 4 mm. in diameter, passed in the anterior interventricular sulcus to the inferior margin, lying by the side of a slightly larger branch from the stem of the left coronary artery. The two vessels ran side by side in the anterior longitudinal furrow, the left one giving a superficial branch and then entering the muscular coat half-way down, while the right artery ran onwards superficially. The wall of both of these vessels was greatly thickened, and the lumen in each reduced to a minute capillary cleft.

Half an inch from its root the diameter of the right coronary artery was 8 mm. At this point the thickening was less pronounced and the lumen wider. The artery continued as a large vessel and gave off numerous branches; a small branch in the epicardial fat along the right margin; a very tortuous branch which ran on the inferior surface an inch from the right margin; a small vessel to the base of the ventricle; at the left portion of the coronary sulcus three branches to the inferior surface of the ventricle, arising close to one another and measuring 2-3 mm. in diameter; finally, the terminal portion of the artery turned downwards in the inferior interventricular sulcus. The distal portion of the artery showed much slighter pathological change, the lumen, though diminished, being distinct.

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The left coronary artery was smaller than the right, and its wall was less affected by pathological change. The external diameter at the root was 6 mm. and the wall not more than 1 mm. thick. It gave off a large branch already mentioned to the anterior interventricular furrow; the wall of this branch was more affected pathologically than the stem of the artery, there being marked intimal thickening in patches near the root, while an inch or two distally the thickening was even more pronounced, involving the whole wall and reducing the lumen to an extremely small

size. The left coronary artery gave a large branch to the left margin of the heart. The wall of this branch too was distinctly thickened, and in the more distal portion so thickened as to reduce the lumen to the smallest dimensions.

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THE CONDITION OF THE HEART IN RELATION
TO THE SYMPTOMS

(1) The first signs of heart impairment occurred in 1908, seventeen years before his death, when he experienced a sharp attack of severe pain, which with our present knowledge would be diagnosed as due to a coronary thrombosis. There is evidence in the heart to confirm this view, for the patch of fibrosis near the apex corresponds to the structural damage which would be caused by such an attack.

Sir Thomas Lewis, who was good enough to send me his opinion after examining the heart, wrote to me as follows:

"Grant and I examined the heart very closely and we are agreed that there are amply sufficient old-standing changes at the apex of the heart to account for the first attack of pain described in his case notes. That attack of pain is strongly suggestive of coronary thrombosis, and the fibrosis at the apex is distributed in a way that also suggests thrombotic obstruction of an apical branch."

(2) The severe atheroma of the coronary arteries and their branches, with diminution of the lumen affords ample cause for the occurrence of attacks of cardiac pain. Both of the arteries were affected and the anterior interventricular branch of each was greatly narrowed.

(3) There were numerous small patches of cicatrization in the substance of the muscular wall of the left ventricle. These patches though smaller were of the same nature as the larger fibrous patch at the apex, which was due to thrombosis of an apical branch. Other arteries to the left ventricle were profoundly altered and their lumen narrowed. This has been shown for example in the marginal artery of the left ventricle. There would therefore appear to have been numerous small thromboses at different times, each of which would be accompanied by symptoms similar to those experienced at the first attack. Several such attacks are recorded and the similarity is brought out in the case history. It is noted, for example, that on many occasions the attacks came on during rest and were quite unrelated to effort.

(4) The presence of numerous small blood vessels on the surface of the heart points to there having been an opening up of small vessels and the establishment of at least a partial anastomotic pathway for the supply of blood to the areas most severely impaired by the attacks of thrombosis. In this connection it may be remarked that during the last few months

of his life between August 1924 and January 1925, Sir James's condition showed slight improvement. I found, for example, that not only could he walk for some distance down Exhibition Road from his home in Albert Hall Mansions, but he was able to walk up that road without distress, though at a slow pace. This improvement doubtless was due to a slight improvement, by anastomosis, in the arterial supply to the heart.

(5) The terminal severe attack of pain and cardiac impairment was associated with the occurrence of the recent infarction found at the apex of the left ventricle.

(6) There was no evidence of any impairment in the valves or in the genetic system of the heart. The impairment was entirely in the muscular wall, brought about by the atheromatous disease of the arteries.

References

Mackenzie, Sir J. (1925): Diseases of the Heart, Ed. 4, London, p. 179.
     (1923): Angina Pectoris, London, p. 176.
     (1922): Reports of the St. Andrews Institute for Clinical Research, London, I, 67.
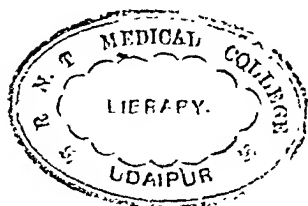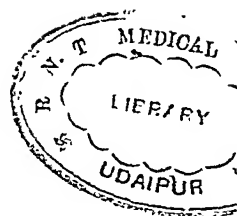
1909

SIR WILLIAM OSLER

DESCRIPTION OF THE SKIN NODULES IN SUBACUTE
BACTERIAL ENDOCARDITIS, LATER TO BE
KNOWN AS OSLER NODES

SIR WILLIAM OSLER

After a crayon portrait by the American artist, John Singer Sargent. Reproduced by permission of the College of Physicians of Philadelphia.
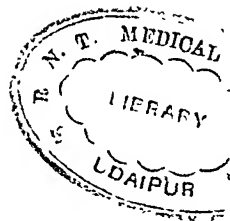
SIR WILLIAM OSLER

(1849-1919)

"I have loved no darkness
Sophisticated no truth
Nursed no delusion
Allowed no fear."

—William Osler.

WILLIAM OSLER was the son of a clergyman, the Reverend Featherstone Lake Osler, and of Ellen Free Pickton Osler. He was born, the sixth son in a family of nine children, in the parsonage at Bond Head, Ontario, near the lower edge of the upper Canadian wilderness, on July 12, 1849.

Young Osler gained his early education at the local grammar school in Dundas, a town of 3,000 people, to which his family had moved in 1857. For some boyish prank Osler was expelled from this school in June of 1864. The next autumn, following in the footsteps of his brothers, he was sent to a boarding school at Barrie. At Barrie, Osler excelled in sports and at one time won the school prize for kicking the football the longest distance. And at Barrie, accompanied by a friend, he swam one and a half miles across Kempenfelt Bay, a feat rendered difficult by the chilliness of the water.

Because Barrie was far from his home in Dundas, Osler, in 1866, was sent to study at the Episcopal school at Weston. Under the influence of the Reverend William A. Johnson, this school had become known as the Trinity College School, and was preparatory school for Trinity University. Osler derived much benefit from his association with Johnson, whom he greatly admired: among other things, he was introduced by Johnson to microscopy. There, also, young Osler became acquainted with Dr. James Bovell, the medical director of Trinity College.

Osler matriculated at Trinity University in the fall of 1867, following his winning of one of the Dixon Prize Scholarships at Weston. During the spring quarter of 1868, Osler indicated his interest in medicine by attending the medical school in the afternoons. In the fall of 1868 he abandoned his course in liberal arts and studied medicine at the Toronto Medical School.

While he was a medical student, Osler published his first paper, "Christmas and the Microscope." It appeared in Hardwicke's "Science-Gossip" for February, 1869. In that paper the young student of microscopy enumerated the living things in a bottle of spring water which he was able to identify with the aid of a microscope. Such was Osler's modest beginning of an extraordinary career in letters.

In 1870 Osler decided to study medicine in Montreal at the McGill Medical School. There he worked under four brilliant men: R. Palmer Howard, Adam H. Wright, Duncan MacCallum, and J. Morley Drake. Three of his teachers had studied under Robert Graves and William Stokes at Edinburgh, and indeed, McGill closely followed the educational methods of the famous Scotch school. Osler was graduated from McGill in 1872, winning a special prize for his thesis.

Osler spent the next two years in postgraduate study in Great Britain. For several months he worked in the laboratory of John Burdon Sanderson at the University College Hospital of London. Thirty-four years later he was destined to succeed Sander-

son as Regius professor of medicine at Oxford. The results of some of his studies in Sanderson's laboratory on hematologic problems were read before the Royal Microscopical Society and subsequently were published in its journal.[1]

For the next few months Osler continued his hematologic studies and under the microscope observed the peculiar globoid bodies now known as blood platelets. Previous investigators had observed these bodies but none of his predecessors actually had seen them in the circulating blood. To Osler belongs the credit of establishing this important contribution to medicine on a firm basis.

Leaving England Osler traveled on the Continent and studied at the medical centers in Berlin and Vienna. He returned to London for a few months and then embarked for his native country. Soon after returning to Canada, Osler accepted an appointment at McGill University as lecturer on the institutes of medicine (1874). In 1876 he was appointed to the newly created post of pathologist at the Montreal General Hospital, and in the spring of 1878 he succeeded Drake as full-time physician in the same hospital.

Osler returned to London for a short visit in 1878 and qualified for and received his membership in the Royal College of Physicians. The same year he attended the meeting of the British Medical Association and there, as Cushing wrote, no doubt became acquainted with Sir T. Grainger Stewart, Sir Jonathan Hutchinson, Sir Clifford Allbutt, Sir William Gairdner, and Sir William Broadbent, who later were to become his staunch friends.

Early in the autumn of 1878, Osler returned to McGill University to resume his medical teaching. He had felt for some time that one of the errors in the practice of medicine was overemphasis on the use of drugs in the treatment of disease. For this reason he believed with Hahnemann that the natural tendency of disease was toward recovery, provided that the patient was decently cared for, properly nursed, and not overdosed. Therefore, when Osler returned as full-time physician to McGill General Hospital he adopted therapeutic methods which were a revelation to the older physicans in the hospital.

In the spring of 1880, Osler, who had been greatly interested in the principles of the physiology of digestion made possible by Beaumont's researches on Alexis St. Martin, desired to perform a necropsy on his body when he learned of St. Martin's demise. It was Osler's intention to preserve St. Martin's stomach and send it to the United States Army Medical Museum in Washington, D. C. The relatives of St. Martin refused Osler the permission for a necropsy. The refusal was a great disappointment to him.

In 1881, Osler and R. Palmer Howard represented McGill University at the meeting of the International Medical Congress held in London under the presidency of Sir James Paget. It was the privilege of those in attendance to hear excellent addresses by Paget, Virchow, John Shaw Billings, Huxley, and Pasteur. Osler profited much from these meetings and probably his contact there with Billings encouraged Osler in several important bibliographic undertakings and in his later interest in medical libraries. Osler read a paper at this congress.

Following another trip to London in 1884, Osler accepted the chair of clinical medicine at the University of Pennsylvania, and he remained in the United States until 1905. In Philadelphia, he made many friends including S. W. Gross, Minis Hays, James Wilson, and Weir Mitchell.

Through much clinical and pathologic study at Montreal, Osler had come to believe that endocarditis was bacterial in origin. In Philadelphia he continued his re-

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[1] Osler, William: Action of certain reagents—atropia, physostigma, and curare—on the colourless blood-corpuscles. Quart. J. Microscopical Sc. 13: 307-309, 1873.

search in this subject and the studies he made there formed the basis for his Goulstonian lectures, which he delivered in London in 1885. In August of that same year Osler, as president of the Canadian Medical Association, gave the annual address at the meeting of the association in Chatham, Western Ontario.

Osler soon was elected to membership in the College of Physicians of Philadelphia and there he made much use of that organization's library, which at that time contained more than 34,000 volumes. He served for five years as a member of the library committee of the College and under his auspices many precious books were added to the library.

In 1889, Osler accepted the offer from the newly opened Johns Hopkins Hospital in Baltimore to serve as physician-in-chief. He assumed his new duties in May, and on him fell the responsibility of organizing the new clinic. Other chiefs-of-staff who were also destined to make Johns Hopkins a famous medical center were William Welch, W. S. Halstead, Henry M. Hurd, and Howard A. Kelly.

In 1891 Osler began to write his great work, "The Principles and Practice of Medicine," first published in 1892. The book filled a decided need and was eagerly received by the medical profession in the United States, Canada, and England. Edition after edition of this popular and important work was published and later, under the editorship of McCrae, it continued in many editions. It is still being published under the editorship of Henry A. Christian and still is the standard textbook in many medical schools.

One of the things that Osler missed in Baltimore was the fine library of the College of Physicians of Philadelphia. In 1891 he volunteered to serve on the library committee of the then almost defunct Medical and Chirurgical Faculty of Maryland, which had a library of a few hundred musty tomes. He aided in the rejuvenation of the faculty and kept his membership on the library committee until his departure for England in 1905, and saw the library grow to the size of 15,000 volumes.

Osler kept his interest in books and libraries throughout his life and contributed financial and moral support to the libraries of McGill, the United States Surgeon General's Office, the College of Physicians of Philadelphia, and to his old preparatory school of Weston, Trinity College School. To many others he made important gifts.

On May 7, 1892, Osler married Grace Revere Gross, the widow of his former friend, Dr. Samuel W. Gross, who had died in April, 1889. Dr. and Mrs. Osler spent the summer of 1892 in England, but even on his honeymoon he could not be kept away from medical meetings.

They returned to Baltimore in August, 1892. On October 4 of that same year, Osler gave an address in Minneapolis on the occasion of the opening of the new medical buildings at the University of Minnesota. The following day he addressed the Minnesota Academy of Medicine choosing as his subject, the "License to Practice."[2]

In December, 1892, Osler received a splendid offer from McGill University, but did not accept it and devoted himself to problems of organization connected with the opening of the new medical school at Johns Hopkins University in Baltimore.

The ensuing years at Johns Hopkins were extremely busy ones for the chief of the medical staff. He was called on countless times to deliver speeches, to write many medical and literary contributions, and to lecture to undergraduate and postgraduate students.

Among the many honors that he won, Osler especially cherished his election, in 1898, to the Royal Society. Election came as a complete surprise to him. It happened that at the time Lord Lister was president of the world-famous body. In the same year Osler became dean of the School of Medicine of Johns Hopkins University.

²Northwest. Lancet 12: 383, 1892.

In the summer of 1898, Osler made another trip to Great Britain. During his stay he was awarded two honorary degrees of Doctor of Laws, one from the University of Aberdeen and one from the University of Edinburgh.

Osler had, for many years, wished to make his home in Great Britain, and had been tempted by the desires of many of his British friends to be a candidate for the chair of medicine at the University of Edinburgh. The chair became vacant in 1900, at which time Osler did become a candidate, but he reconsidered and withdrew his application. In August, 1904, however, being offered the position of Regius professor of medicine at the University of Oxford, he accepted.

Osler assumed his new office in the latter part of May, 1905. His first official duty was to act as a curator, ex officio, of the Bodleian Library, a task, we may assume, from which he derived much pleasure. In a special convocation in June, 1905, he received from Oxford University the degree, Doctor of Science.

On October 18, 1906, Osler gave the annual Harveian oration. He chose for his subject, ''The Growth of Truth as Illustrated in the Discovery of the Circulation of the Blood.''

In 1907 appeared the first three volumes of Osler's ''System of Modern Medicine,'' which was to become an epoch-making contribution to medicine.

In 1908 he was chosen to give the Linacre lecture. This he delivered on May 6 at St. John's College, Cambridge, choosing the life of Thomas Linacre as his subject. On June 12 he read before the Association of Physicians of Great Britain and Ireland his classic account of ''Chronic Infectious Endocarditis.'' This contains the description of the ''Osler nodes.'' We are presenting this account to our readers.

In the spring of 1910, Osler delivered the famous Lumleian lectures before the Royal College of Physicians. He chose angina pectoris as the subject of his address. In 1911 at the Coronation of King George, Osler was created a baronet.

Osler made his last visit to the United States in 1913. To students of medical history this was an auspicious occasion, for in that year he gave a course of lectures on ''The Evolution of Modern Medicine,'' the Silliman Lectures, at Yale University. These were later published in book form and constitute one of his best works. That year, also, he served as president of the medical section of the International Medical Congress at its meeting in London.

In 1914 Osler was elected president of the Bibliographical Society. He chose as the subject of his address ''The Earliest Printed Medical Books.'' This was published as a preface, nearly four years after his death, to his important bibliographic study, ''Incunabula Medica'' (1923).

During the World War, Osler served as a civilian member of the committees at the War Office. He also served on the Committee for the Medical History of the War and on the War Reports Committee. He held the position of honorary colonel in the army and helped greatly in building up the morale of the British, Canadian, and American soldiers who returned wounded from the front.

It had been Osler's philosophy to accept the sorrows and the joys of the world with equanimity, but the death of his only son, Revere, who had been severely wounded in France, occurred in August, 1917, and was a blow very hard for him to sustain.

In July, 1919, Osler celebrated his seventieth birthday. He had suffered many attacks of bronchopneumonia, which left him very weak. The exertions of his last few years, the heart-breaking loss of his son, and the heavy strain of the war all contributed to the undermining of his health. His final illness was protracted influenza, to which he succumbed on December 29, 1919.

CHRONIC INFECTIOUS ENDOCARDITIS*

By

WILLIAM OSLER

[Description of Osler Nodes]

A N ENDOCARDITIS with fever as its only symptom may be prolonged for weeks or months under many different circumstances. Following rheumatic fever in a child an endocardial complication may keep up a temperature of from 100° to 101° for several months, during which time there may be no other symptoms and the general condition may remain fairly good. In chronic valvular disease in the stage of broken compensation slight irregular fever may persist for months, associated with the presence of fresh endocarditis. As a rule, the form of endocarditis to which we give the term infective, septic, or ulcerative runs its course under three months. That occasional instances were characterized by a very protracted course was noted by Wilks, Bristowe, Coupland, and Lancereaux. In my Goulstonian Lectures, 1885, I stated that this type had the following characteristics: The fever was irregular and intermittent, resembling ague; the cold, hot, and sweating stages might succeed each other with great regularity; in the intervals fever might be absent; two or three paroxysms could occur in the course of a day. In many of the instances the disease was prolonged to three or four months, and I give the notes of a case of Bristowe's, in which the condition persisted for five months. The recurring chills usually led to the diagnosis of malaria and also gave rise to the opinion widely held, particularly by French writers, that ulcerative endocarditis could be caused by this disease. The cases to which I wish to call attention in this communication are of this chronic character, not marked specially by chills, but by a protracted fever, often not very high but from four to twelve months' duration. At the time of the delivery of the Goulstonian Lectures I had not seen a case of this type. In the past twenty years I have seen ten cases of this form, two of which I have already reported (*Practitioner*, 1893).

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It has long been recognized that malignant endocarditis is really an acute septicaemia with localization on the endocardium, but the symptoms are not necessarily due to the local lesion. The clinical picture is a

septicaemia sometimes of a typhoid type, sometimes like a pyaemia—then again with predominant meningeal symptoms, occasionally with pronounced cardiac features. The pneumococcic, the gonorrheal, and the streptococcic forms present, as a rule, a picture in which the heart-symptoms are in the background. Cases of infection with these organisms may run an identical course without any endocarditis. On the other hand, there is a large group of cases in which the endocarditis plays a more important role and the vegetations and ulcerations appear to be directly responsible for the fever and the associated symptoms. As a rule, the valves involved are already the seat of a sclerotic change. The source of the infection is rarely to be determined. Thus, in only one of the series here reported was there an external lesion. The patients in this series were all adults, five women and five men. In six there was a past history of rheumatic fever; eight had old mitral lesions, two aortic, well compensated, and not giving any trouble at the time of the onset of the symptoms. It was not always possible to get a definite history of how the attacks began. In five of the cases there were chills and fever, mistaken for malaria. Cough and loss of weight in some cases suggested tuberculosis. The slight fever without any localizing symptoms may raise the suspicion of typhoid fever. In my series these have been the three diseases the diagnosis of which has been suggested. Once established the fever becomes the dominant, and for months may be the only, symptom. This is the most striking peculiarity of the cases. Week after week, month after month, the daily rise of one and a half or two degrees may be the only indication there is of an existing mischief. In Case I, in which the fever lasted for thirteen months, the patient's sister, a trained nurse, had decorated the room with yards of the temperature charts; fever with an occasional sweat were the only symptoms. The appetite remained good and she lost very little in weight. There were no embolic features and from month to month there were few, if any, changes in the cardiac condition. In this very protracted form chills are not nearly so common as in the more acute cases, nor is the fever so high, not often reaching above 102.5° or 103°. It is of a remittent type, not falling to normal at any period of the day. With the occurrence of a chill the temperature may rise to 104° or 105°, but in none of the cases was there the type of fever in which the paroxysms recur with great regularity—quotidian or tertian, as we see so often in the acute forms of ulcerative endocarditis. Another peculiarity is the occurrence of periods of apyrexia, usually towards the end, but in one or two of the cases there were afebrile interludes which gave deceptive promise of recovery. It is well recognized now that fever is not an invariable accompaniment of endocarditis. Following pneumonia there may be for months a slight toxaemia with little or no fever in connexion with a patch of endocarditis.

The cardiac features in this group are usually well marked, but as a rule there are no symptoms. The patients complain neither of palpitation

nor of pain. There is no dyspnoea except towards the close, and in no case did dropsy occur. In eight of the ten cases there were the well-marked physical signs of a mitral lesion and the associated slight enlargement of the heart. In only six cases was there marked hypertrophy and dilatation. In two of the cases there was aortic insufficiency. One of the most striking circumstances is the very slight change in the character of the heart murmur in spite of the fact of most extensive vegetations and alterations in the valves. Thus in the case of Dr R T., with the condition of whose heart I had been familiar for fourteen years, the comparison between my first examination in 1889 and that in 1893 showed very little change beyond the slightly greater dislocation outwards of the apex beat. In several of the cases the absence of any change in the character of the heart murmur and the remarkably quiet, negative state of the organ were urged strongly against the existence of endocarditis. It is rather remarkable, considering the anatomical changes, that so little alteration may occur in the physical signs. In Case VI, Dr. B. T., the murmur of aortic insufficiency became more intense towards the close, but in no instance was there the development under observation of alterations in the physical signs such as are sometimes seen in acute ulcerative endocarditis.

Embolism, to cause symptoms, occurred in four cases of the series—in Cases III, IV, and IX in the brain with haemiplegia, Case VIII in the retinal arteries and in the spleen and kidneys. This is in striking contrast to the frequency of this complication in the more acute types of endocarditis.

One of the most interesting features of the disease and one to which very little attention has been paid is the occurrence of ephemeral spots of a painful nodular erythema, chiefly in the skin of the hands and feet, the *nodosités cutanées éphémères* of the French. My attention was first called to these in the patient of Dr. Mullen of Hamilton, whose description is admirable: "The spots came out at intervals as small swollen areas, some the size of a pea, others a centimetre and a half in diameter, raised, red, with a whitish point in the centre. I have known them to pass away in a few hours, but more commonly they last for a day, or even longer. The commonest situation is near the tip of the finger, which may be slightly swollen." Spots of this character occurred in seven of the cases and in three at least they were of importance in determining the diagnosis. Thus in the case of Dr. Carroll, the well-known American Army Surgeon, the collaborator with Dr. Reed in the brilliant work upon yellow fever, the presence of these spots appeared to me to clinch the diagnosis. They are not beneath but in the skin and they are not unlike an ordinary wheal of urticaria. The pads of the fingers and toes, the thenar and hyperthenar eminences, the sides of the fingers, and the skin of the lower part of the arm are the most common localities. In one case they were present in the skin of the flank. I have never seen them haemorrhagic, but always erythematous, sometimes of a very vivid pink hue, with a slightly opaque centre.

The diagnosis in this group of cases may offer great difficulties. For weeks, indeed for several months, there may be only fever, and unless there have been special features pointing to the heart, such as the development of a diastolic murmur or the great intensification of a mitral bruit, it may be impossible to settle the diagnosis. There are, indeed, cases in which from beginning to close no heart murmur has been present. By far the most suggestive features are: (1) a knowledge of the existence of an old valve lesion. This was present in every one of my series. (2) The occurrence of embolic features, sudden swelling of the spleen, with friction in the left flank, sudden attack of haematuria, embolism of the retinal arteries, hemiplegia or the blocking of a vessel in one of the limbs. (3) The onset of special skin symptoms, purpura, and more particularly the painful erythematous nodules to which I have referred. Present in seven of the ten cases, these are of definite diagnostic import. They are in all probability caused by minute emboli. (4) The progressive cardiac changes, the gradual increase in the dilatation of the heart, the marked change in the character of a mitral murmur, the onset of a loud rasping tricuspid murmur, or the development under observation of an aortic diastolic bruit.

With carefully made blood-cultures one should now be able to determine the presence of the septicaemia. This was easily done in three of my more recent cases. An onset with chills and fever and slight swelling of the spleen almost always leads to the diagnosis of malaria, more particularly in regions in which this disease prevails, but in not one of my cases was there any difficulty in excluding this by careful microscopical examination of the blood. It was not always possible to convince the physician. With slight cough tuberculosis may be suspected, as happened in two or three cases of my series. For many weeks the patient may present nothing but a pyrexia, of doubtful origin, or a cryptogenetic septicaemia and as he may look very well and may feel very well, and there are no special symptoms, and with a heart-condition that may have remained unchanged for years, it is not easy to reach a positive diagnosis. The blood-cultures and the presence of the painful erythematous nodules and the occurrence of embolism furnish the most important aids.

The anatomical condition in these cases is quite unlike that of the ordinary ulcerative endocarditis. In the three specimens I have had an opportunity of studying there was no actual ulceration, but large proliferative vegetations, firm and hard, greyish yellow in colour, projected from the endocardium of the valves like large condylomata, encrusting the chordae tendineae and extending to the endocardium of the auricle. The condition is quite unlike the globose vegetations of the pneumococcal and gonorrhoeal endocarditis or the superficial ulcerative erosions of the acute septic cases.

The organisms responsible for this condition have been carefully studied. In my series cultures were made in six cases. In three they were negative. In two streptococci were present, in one a staphylococcus.

While, as a rule, this condition is much more commonly caused by the streptococcus other organisms may be present. Thus Fraenkel has reported one instance of a pneumococcus endocarditis persisting for nearly six months (*Deutsche med. Woch.*, 1900). Of sixteen cases of this chronic form, the clinical course of which extended from four to eight months, Harbitz (*Deutsche med. Woch.*, 1899) found pneumococci in four, streptococci in nine, and in eight other micro-organisms. Lenhartz (*Deutsche med. Woch.*, 1901), who has reported sixteen cases with a duration of from three to seven months, found staphylococci and streptococci the common organisms, the pneumococcus once and the gonococcus once. In the majority of cases it seems to be a mild streptococcus infection, possibly by a special form. Possibly in some instances there may be a special resistance on the part of the host, but these are points which must be settled by future investigations. These are cases in which the possibility of successful vaccine treatment should be considered. It was tried in two cases of my series, but in both rather late, and in neither did it seem to have special influence. Horder has treated a case of this chronic type with a vaccine prepared from the patient's organism, but without success. The results in the acute forms are discussed by him in the *Practitioner*, May, 1908.

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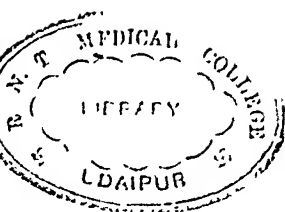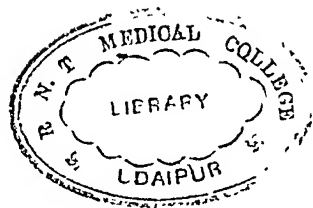
1912

JAMES B. HERRICK

DESCRIPTION OF CORONARY THROMBOSIS

JAMES BRYAN HERRICK

(Courtesy Central Interurban Clinical Club.)

JAMES BRYAN HERRICK

(1861-1954)

"Men succeed because of native ability and in small measure because of chance, but chiefly through hard work, through a knowledge of their special vocational subject and through their ability to apply this knowledge."   —J. B. Herrick, in his essay, "N. S. Davis."

*J*AMES BRYAN HERRICK, a living member of the great company of classic cardiologists, whose outstanding contribution to this field we are reprinting herein, was born on August 11, 1861, in Oak Park, Illinois, the son of Origen White Herrick and Dora E. Herrick. His maternal grandfather operated a saw mill on the Desplaines River, and his mother, the former Dora E. Kettlestrings, who was also born in Oak Park was (in 1935) the oldest native daughter of that village. Her father immigrated to the United States from England in 1833, traveled westward in a covered wagon and settled on a homestead on the site which is now Oak Park.

James Herrick attended the Oak Park High School and the Rock River Seminary at Mount Morris, Illinois. He received his collegiate training at the University of Michigan, where he was graduated with the degree of Bachelor of Arts in 1882. At Michigan, young Herrick came under the influence of Moses Coit Tyler, professor of English literature. Tyler was not only an authority on American literature, having published the best history of American literature of the colonial and revolutionary periods, but also was a Chaucerian scholar of the highest order. Under Tyler's influence, Herrick was instilled with a profound appreciation of Chaucer that has endured.[1]

Herrick began the study of medicine in 1886, choosing Rush Medical College as his school. He was graduated from Rush in 1888 with the degree of Doctor of Medicine, and he interned at the Cook County Hospital. In 1889 he married Zellah P. Davis, of his native town of Oak Park.

In addition to making important contributions to medical literature at an early date, Herrick found time to pursue literary activities. He also acted as an officer of the board of the Lewis Institute and the McCormick Memorial Institute. At this writing, he is secretary of the board of trustees of the Lewis Institute and is president of the McCormick Memorial Institute in Chicago.

One year (1889) after his graduation from Rush, Herrick contributed three articles to medical literature. The first of these was "A Case of Hemophilia Neonatorum." This appeared in the first volume of the "North American Practitioner." By a typographical error, the article was credited to James B. Henrick. In the same volume Herrick reported on traumatic rupture of the bladder. In the "Western Medical Reporter" he described an operation for sacro-iliac tuberculosis. From 1889 until 1935, according to Holmes, there have been only five individual years in which Herrick did not contribute to scientific literature. In 1896 he published eleven articles, including three on cardiovascular disease and two on anemia.

In 1904 Herrick made an original observation in hematology. At that time a negro who had a sore on his ankle and evidences of previous scarring presented himself for treatment. In making a hematologic examination Herrick discovered that the blood of this patient showed numerous elongated or sickle-shaped red cells. Although the discovery was made in 1904 and was confirmed in 1906 by E. E. Irons, Herrick waited until 1910 to publish it.[2] In 1910 also appeared his first article on angina pectoris—

In 1910 also appeared his recent article, Why I Read Chaucer at

[1] Herrick's appreciation is well expressed in his recent article, Why I Read Chaucer at 70, Ann. Med. Hist. 5: 62-72, 1933.
[2] Herrick, J. B.: Peculiar elongated and sickle-shape red blood corpuscles in a case of severe anemia, Arch. Int. Med. 6: 517-521, 1910.

a disease on which he later made many scholarly contributions, and with which his name probably will be associated in the future.

Herrick worked hard to make the profession recognize the importance of coronary thrombosis, and in 1912, the "Journal of the American Medical Association" published his classical account of this condition in a paper entitled "Clinical Features of Sudden Obstruction of the Coronary Arteries." Besides giving to the medical world by far the best extant description of this disease, Herrick showed that sudden obstruction of a coronary artery is not necessarily fatal. We consider it a special privilege to reprint in full this historic classic. It is of significance to note, in this connection, that in January, 1939, Herrick[3] pointed out that Robert Adams about 100 years ago came very close to the discovery of coronary thrombosis. Adam Hammer described the first case of coronary thrombosis with correct diagnosis ante mortem in 1878,[4] and the first completed description of the disease was published in 1910 by Obrastzow and Straschesko.[5]

In 1918 Herrick and Nuzum[6] made the first direct reference to the occurrence of anginal pain among patients having severe anemia.

Since 1918 Herrick has made many contributions to the literature of medicine. The reader is referred to the bibliography of 135 articles by Herrick compiled by Elizabeth Carr, librarian of the Northwestern University Medical School. This constitutes an appendix to William H. Holmes' appreciative study of Herrick, from which study the data in the present brief biographic account were, in part, obtained.

Dr. Herrick has received many honors. He is past president of the Chicago Pathological Society; he was the first (1915) president of the Chicago Society of Internal Medicine; he has served as president (1925) of the Institute of Medicine of Chicago and of the Association of American Physicians (1923), and he was president of the American Heart Association in 1927. In 1938 he was again honored by the Association of American Physicians by being elected vice-president of that distinguished group of internists. His services with the McCormick Memorial Institute and the Lewis Institute have already been mentioned. As this is written (1940), he is president of the Congress of American Physicians and Surgeons.

The University of Michigan has twice honored her distinguished son: in 1907, with an honorary degree of Master of Arts, and in 1932, with the degree of Doctor of Laws.

Dr. Herrick has been affiliated with many institutions in many professional capacities. He was instructor in medicine at Rush Medical College from 1890 to 1894, assistant professor of medicine at the same school from 1894 to 1900, and professor of medicine from 1900 to 1926. Since 1926 he has been emeritus professor in the same college. He also was professor of the theory and practice of medicine and professor of materia medica and therapy at the old Northwestern University Women's Medical School, which became extinct in 1902. Since 1898 he has been attending physician to the Presbyterian Hospital in Chicago. He is an honorary fellow of the New York Academy of Medicine, and in 1931 was selected to be the lecturer of the Harvey Society of that institution. In 1930 he was awarded the George M. Kober Medal conferred by the Association of American Physicians. In 1939, at the ninetieth annual meeting of the American Medical Association, Dr. Herrick received the second Distinguished Service Medal to be awarded by that association, the first having been awarded in 1938 to Dr. Rudolph Matas of New Orleans.[a]

[3] Herrick, J. B.: Robert Adams; surgeon, and his contributions to cardiology. Ann. Med. Hist. 1: 45-49 (Jan.) 1939.

[4] Hammer, Adam: Ein Fall von thrombotischem Verschlusse einer der Kranzarterie des Herzens. Am Krankenbette konstatirt. Wien. med. Wchnschr. 28: 97-102, 1878.

[5] Obrastzow, W. P. and Straschesko, N. D.: Zur Kenntnis der Thrombose der Koronararterien des Herzens. Ztschr. f. klin. Med. 71: 116-132, 1910.

[6] Herrick, J. B. and Nuzum, F. R.: Angina pectoris, J. A. M. A. 70: 67-70, 1918.

[a] [Dr. Herrick died in 1954.]

CLINICAL FEATURES OF SUDDEN OBSTRUCTION
OF THE CORONARY ARTERIES*

By

JAMES B. HERRICK, M. D.

Chicago

OBSTRUCTION of a coronary artery or any of its large branches has long been regarded as a serious accident. Several events contributed toward the prevalence of the view that this condition was almost always suddenly fatal. Parry's writings on angina pectoris and its relation to coronary disease, Jenner's observations on the same condition centering about John Hunter's case, Thorvaldsen's tragic death in the theater in Copenhagen with the finding of a plugged coronary, sharply attracted attention to the relation between the coronary and sudden death. In Germany Cohnheim supported the views of Hyrtl and Henle as to lack of considerable anastomosis, and as late as 1881 lent the influence of his name to the doctrine that the coronary arteries were end-arteries; his Leipsic necropsy experience, as well as experiments on dogs, forced him to conclude that the sudden occlusion of one of these vessels or of one of the larger branches, such as the ramus descendens of the left coronary, meant death within a few minutes. Others emphasized the same view.

No one at all familiar with the clinical, pathologic or experimental features of cardiac disease can question the importance of the coronaries. The influence of sclerosis of these vessels in the way of producing anemic necrosis and fibrosis of the myocardium, with such possible results as aneurysm, rupture or dilatation of the heart, is well known. So also is the relation of the coronaries to many cases of angina pectoris, and to cardiac disturbances rather indefinitely classed as chronic myocarditis, cardiac irregularities, etc. It must be admitted, also, that the reputation of the descending branch of the left coronary as the artery of sudden death is not undeserved.

But there are reasons for believing that even large branches of the coronary arteries may be occluded—at times acutely occluded—without resulting death, at least without death in the immediate future. Even the main trunk may at times be obstructed and the patient live. It is the main object of this paper to present a few facts along this line, and particularly to describe some of the clinical manifestations of sudden yet not immediately fatal cases of coronary obstruction.

Before presenting the clinical features of coronary obstruction, it may be well to consider certain facts that go to prove that sudden obstruction is not necessarily fatal. Such proof is afforded by a study of the anatomy of the normal as well as of the diseased heart, by animal experiment and by bedside experience.

The coronaries are not so strictly end-arteries, i.e., with merely capillary anastomoses, as Cohnheim and others thought. By careful dissections, by injection of one artery from another, by skiagraphs of injected arteries and by direct inspection of hearts made translucent by special methods, there is proof of an anatomic anastomosis that is by no means negligible.

Jamin and Merkel's* beautiful stereoscopic skiagrams show the remarkably rich blood-supply of the heart, with occasional anastomoses between vessels of considerable size. The possibility of injection of the coronary artery from the other is admitted even by those who deny that such injection proves more than a capillary non-functioning anastomosis. Amenomiya,[1] by injecting hearts of young persons, showed naked-eye anastomoses in the subepicardial tissue. He feels that Hirsch and Spalteholz[2] have nearly cleared up the question as to the relation between the heart muscle and disease of the coronary artery from the anatomic standpoint. Hirsch says that in dogs the anastomosing vessels are functionally competent, and Spalteholz says that in man the vessels are nearly the same as in dogs, rich in anastomoses even in those of considerable caliber. The latter investigator, by a method of injection and treatment of the heart so as to make the muscle transparent, shows to the naked eye that there are anastomoses of considerable size.

Among others who are on record as believing that there are non-negligible anastomoses may be mentioned Haller, Huchard, Orth, Michaelis, Langer, Legg, West. All recognize, however, that there are individual differences, and also that though the heart may show rich anastomoses, these are not necessarily functional, i.e., that an artery which anatomically is not a terminal artery may yet be such functionally.

But there is proof not only of anatomic connection between the two coronaries, but that in certain instances, at least, such connection is of functional value. Experiments on lower animals and the clinical experiment of disease of the coronaries with autopsy findings show this.

Much of the earlier experimental work on the lower animals, obstructing the coronary arteries by ligatures, clamps or artificial emboli, gave promptly fatal result. Among those who worked along this line and

*Jamin and Merkel: Die Koronararterien des menschlichen Herzens in stereoskopischen Röntgenbildern, Jena, 1907. Extensive bibliographies are contained in the articles by Thorel (Lubarsch-Ostertag's Ergebnisse, ix, Abt. 1), and in Amenomiya (Virchows Arch. f. rath. Anat., 1910, cxcix, 187). I repeat only some of the more important references and add new ones

[1]Amenomiya: Ueber die Beziehungen zwischen Koronararterien und Papillarmuskeln im Herzen, Virchows Arch. f. path. Anat., 1910, cxcix, 187.

[2]Hirsch and Spalteholz: Koronararterien und Herzmuskel, Deutsch. med. Wchnschr., 1907, No. 20.

reached these conclusions may be mentioned Erichsen (1842), Panum (1862), von Bezold, Samuelson (1880), Cohnheim and Schulthess-Rechberg (1881), G. Sée, Rochefontaine and Roussy (1881), Bettelheim (1892), Kronecker, and, to some extent, Michaelis. The work of Cohnheim[3] attracted particular attention and his conclusions as to end-arteries, irreparable injury, and cessation of the beat of both sides of the heart within two minutes from the time of shutting off the coronary circulation confirmed and elaborated the conclusions of the earlier experimenters, and was in turn confirmed by the French writers just named, by Bettelheim and others.

But soon dissent was heard from various quarters as to many of Cohnheim's results, and among other things as to the sudden death following the ligations. Michaelis found that the injury from ligation in rabbits was not so serious or irreparable as in dogs. Fenoglio and Drouguell, in 1888, found that some dogs might live. Porter showed that after ligation of one or two large branches of the coronary artery a dog might live hours or days. More than half his animals lived after ligation of the descending branch of the left coronary. Von Frey, at the Congress for Internal Medicine in 1891, said that he doubted the sudden stopping of the heart as Cohnheim taught; he believed that clearly the greater weight should attach to those observations in which the ligation was borne without harm; and that the stopping of the heart was not a necessary consequence of the obstruction of a large coronary branch. Hirsch in eight dogs and two apes had no sudden deaths from ligation. Bickel,[4] under Orth and Amenomiya, had a dog live nineteen days after the ligation of the descending branch of the left coronary; he killed two dogs, one on the eighth and the other on the seventeenth day after ligation. Kölster ligated smaller branches; his dogs lived, and when killed at intervals of several weeks showed the progressive changes of fibrosis of the myocardium. Imperfect technic, by which damage was done to the heart muscle and pneumothorax produced, is offered as a partial explanation, at least, for the more rapidly fatal results obtained by Cohnheim and others. Miller and Matthews[5] call attention to the better results where ether as an anesthetic is employed rather than curare or other drug. With ether they were able to ligate large branches, many of their dogs living several weeks.

Experimentally, then sudden death, even late death, is not a necessary consequence of obstruction of even large branches, such as the descending branch of a coronary artery.

There are numerous autopsy observations, frequently with helpful clinical history, that show directly or by inference the existence of

[3]Cohnheim and Schulthess-Rechberg: Ueber die Folgen der Kranzarterienverschliessung für das Herz, Virchows Arch. f. path. Anat., 1881, lxxxv, 503.
[4]Cited by Amenomiya (See Note 2).
[5]Miller and Matthews: Effect on the Heart of Experimental Obstruction of the Left Coronary Artery, Arch. Int. Med., June, 1909, p. 476.

efficient anastomoses, and the ability of the heart at times to survive the obstruction of a coronary or some large branch. Some of these instructive cases may be mentioned. Pagenstecher, on account of an accident, ligated the descending branch of the left coronary artery and the patient lived five days. Thorel has seen hearts with complete obstruction of the artery, with fibrous or calcified myocardium, and yet no symptoms during life, the patient dying of some other disease. I have seen the descending branch completely occluded with an extensive fibrous area in the interventricular septum and at the apex, the latter aneurysmally dilated, where the process was clearly one of long standing. West[6] cites several cases in which at autopsy complete obstruction of one coronary was found, yet the patients had long survived this serious lesion.

Chiari, in a 32-year-old nephritic, found a sclerosed right coronary plugged by a thrombus, with resulting scattered patches of myomalacia cordis in the areas supplied by this artery. A portion of this thrombus had become detached and had embolically plugged the left coronary, resulting in sudden death. From the symptoms and the autopsy findings the thrombus in the right artery had formed at least two days before. The fact that the softened patches in the myocardium were scattered, with normal tissue between, and that the heart functionated fairly well until the left artery also was obstructed, leads Chiari to infer that anastomoses must exist between the right and left coronaries. Merkel[7] drew the same inference as to anastomoses from the patchy character of the lesions in the heart of a woman of 76 years, there being normal muscle between the softened areas. The left coronary was the seat of the obstruction. He also saw in a man of 37 the left coronary closed, with nourishment through the right artery. Dock[8] in a case of gradual occlusion of the right coronary artery was able to demonstrate a direct opening of the finer branches of the left coronary into the end of the right.

Spalteholz says that we all know cases of stoppage of large vessels without large infarcts resulting. Recklinghausen and Fujinami found this condition in man, as Hirsch had in dogs and monkeys; i.e., smaller infarcts than the distribution of the vessel would lead one to expect. Galli saw complete closure of the entrance to the right coronary artery yet no change in the myocardium. By injection he found a round-about anastomosis between the right and left coronary arteries. Samuelson cites the case of a patient living five hours after obstruction, Huber one of a patient living several days. Aschoff and Tawara[9] saw a patient live fourteen days, "with nearly complete infarction of the parietal wall of

[6]West: Tr. Path Soc., London, 1882, xxxiv, 67.
[7]Merkel, H.: Ueber den Verschluss der Kranzarterien des Herzens, Festschrift für Rosenthal, Leipsic, 1906.
[8]Dock, George: Notes on the Coronary Arteries, Ann Arbor, 1896.
[9]Aschoff and Tawara: Die heutige Lehre von den pathologisch-anatomischen Grundlagen der Herzschwäche, Jena, 1906, p. 56.

the left ventricle." In several cases of angina pectoris cited by Huchard[10] the patients lived many hours after the onset of the final attack, which autopsy showed was due to a thrombotic closure of an artery. Osler refers to the fact that the patient may live for some time after obstruction. Krehl expressly states that in man the more or less sudden occlusion of an entire coronary artery, or at least a large branch, such as the descending branch, is compatible with a continuance of life.

One may conclude, therefore, from a consideration of the clinical histories of numerous cases in which there has been careful autopsy control, from animal experiments and from anatomic study, that there is no inherent reason why the stoppage of a large branch of a coronary artery, or even of a main trunk, must of necessity cause sudden death. Rather may it be concluded that while sudden death often does occur, yet at times it is postponed for several hours or even days, and in some instances a complete, i.e., functionally complete, recovery ensues.

The clinical manifestations of coronary obstruction will evidently vary greatly, depending on the size, location and number of vessels occluded. The symptoms and end-results must also be influenced by blood-pressure, by the condition of the myocardium not immediately affected by the obstruction, and by the ability of the remaining vessels properly to carry on their work, as determined by their health or disease. No simple picture of the condition can, therefore, be drawn. All attempts at dividing these clinical manifestations into groups must be artificial and more or less imperfect. Yet such an attempt is not without value, as it enables one the better to understand the gravity of an obstructive accident, to differentiate it from other conditions presenting somewhat similar symptoms, and to employ a more rational therapy that may, to a slight extent at least, be more efficient.

The variations in the results are to be accounted for in part by variations in the freedom with which anastomosing branches occur. Presumably, too, symptoms will vary with the vessels or branches occluded. It is conceivable that with occlusion of the right coronary the symptoms might be different from those following obstruction of the left artery; systemic edema might be a consequence of the former condition and pulmonary edema of the latter. These points are, however, by no means settled either by experimental or clinical observation. The condition of the remaining vessels as to patency and presence of sclerosis must play an important part in deciding how much they are capable of doing in the way of compensatory nutrition to the anemic myocardium; the strength of the heart itself, as determined, perhaps, by old valvular or myocardial disease, would also have its influence. And presumably a sudden overwhelming obstruction, with comparatively normal vessels, would be followed by a profounder shock than the gradual narrowing of a lumen

[10]Huchard: Traité clinique des maladies du coeur, second edition, p 560.

through sclerosis which has accustomed the heart to this pathologic condition and has perhaps caused collateral circulation through neighboring or anastomosing vessels to be compensatorily increased. The influence of the vessels of Thebesius is also not to be overlooked in this connection; compensatory circulation through these accessory channels may be of considerable importance in nourishing areas of heart muscle poorly supplied by sclerotic or obstructed arteries.

Attempts to group these cases of coronary obstruction according to clinical manifestations must be more or less unsatisfactory, yet, imperfect as the groups are, the cases may be roughly classified.

One group will include cases in which death is sudden, seemingly instantaneous and perhaps painless. Krehl[11] has emphasized the peculiarities of the sudden death of this type, the lack of terminal respiratory agony, of distortion of the features, of muscular contractions.

A second group includes those cases in which the attack is anginal, the pain severe, the shock profound and death follows in a few minutes or several minutes at the most.

In a third group may be placed non-fatal cases with mild symptoms. Slight anginal attacks without the ordinary causes (such as walking), perhaps some of the stitch pains in the precordia, may well be due to obstruction of small coronary twigs. Such an interpretation of these phenomena is, however, only a surmise based on the fact that other causes for the pains are lacking and that the patchy fibrosis of the myocardium that is later found at autopsy may have originated in obstruction of the sclerotic vessels; and such obstruction in small vessels may well have produced symptoms differing chiefly in degree from those caused by obstruction of larger arteries of the heart.

In a fourth group are the cases in which the symptoms are severe, are distinctive enough to enable them to be recognized as cardiac, and in which the accident is usually fatal, but not immediately, and perhaps not necessarily so. It is to the clinical features of this group that attention is directed in what follows.

By way of introduction, I give in outline the history of a case, experience with which acutely attracted my attention to this subject.

CASE 1.—*History.*—A man, aged 55, supposedly in good health, was seized an hour after a moderately full meal with severe pain in the lower precordial region. He was nauseated and, believing that something he had just eaten had disagreed with him, he induced vomiting by tickling his throat. The pain continued, however, and his physician was called, who found him cold, nauseated, with small rapid pulse, and suffering extreme pain. The stomach was washed out and morphine given hypodermically. The pain did not cease until three hours had passed. From this time the patient remained in bed, free from pain, but the pulse continued rapid and small, and numerous râles appeared in the chest. When I saw him twelve hours from the painful attack his mind was clear and

[11]Krehl: Der Verschluss der Kranzarterien; in Nothnagel's System, xv, 369.

calm; a moderate cyanosis and a mild dyspnea, were present. The chest was full of fine and coarse moist râles; there was a running, feeble pulse of 140. The heart tones were very faint and there was a most startling and confusing hyperresonance over the chest, the area of heart dulness being entirely obscured. The abdomen was tympanitic. The urine was scanty, of high specific gravity, and contained a small amount of albumin and a few casts. The temperature was subnormal, later going to 99° F Occasionally there was nausea and twice a sudden projectile vomiting of considerable fluid material. This condition remained with slight variations up to the time of death, fifty-two hours after the onset of the pain, though at one time the râles seemed nearly to have disappeared A few hours before death, the patient described a slight pain in the heart region, but said it did not amount to much. A remarkable circumstance, and one that occasioned surprise in those who saw the patient and who realized from the almost imperceptible pulse and the feeble heart tones how weak the heart must be, was the fact that he frequently indulged in active muscular effort without evident harm. He rolled vigorously from side to side in the bed, sat suddenly bolt upright, or reached out to take things from the table near by, and once, feeling a sudden nausea, he jumped out of bed, dodged the nurse and ran into the bathroom, where he vomited; and yet seemed none the worse for these exertions.

Necropsy (Dr. Hektoen).—The heart was of normal size, but both coronary arteries were markedly sclerotic, with calcareous districts and narrowing of the lumen. A short distance from its origin the left coronary artery was completely obliterated by a red thrombus that had formed at a point of great narrowing. The wall of the left ventricle showed well-marked areas of yellowish and reddish softening, especially extensive in the interventricular septum. At the very apex the muscle was decidedly softer than elsewhere. The beginning of the aorta showed a few yellowish spots, these areas becoming less marked as the descending part was reached. An acute fibrinous pericardial deposit, which showed no bacteria in smears, was found over the left ventricle. (The pericarditis probably explains the slighter pain complained of a few hours before death.) There was marked edema of the lungs. In other respects the anatomic findings were those of health.

A colleague personally related to me the case of a man of 60 who, three days after a severe anginal seizure, felt well enough to walk on the street, though with some dyspnea. He died suddenly on the fifth day. The obstruction in the left coronary, and the muscular softening found at autopsy were similar to those in the case just described.

Since my attention has been called to this condition, I have seen five other cases that I am convinced were instances of coronary thrombosis, the patients living many hours after the accident, though no autopsy control confirms this opinion. All were men beyond 50. In all there was some evidence of peripheral arteriosclerosis; all had had previous anginal attacks. In all the final attack was described as the severest and most prolonged in the experience of the patient. Morphin alone had given relief. In all the sudden development of a weak pulse, with feeble

cardiac tones, was a striking feature; the pulse was generally rapid. Dyspnea and cyanosis varied in degree. Râles, moist and dry, were usually present. Emphysema was present to a moderate degree in two of the five. Only one patient left his bed after the attack. His pulse showed great improvement as to quality and rate, though dyspnea, râles, edema of the legs, albumin, increased area of cardiac dulness, etc., showed failure of the heart muscle. From the time of the seizure, i.e., the time of the obstruction, to death was in one case three days, in one seven, in two twelve, and in one twenty days.

One of these cases is, it seems to me, a typical one of this sort and, though necropsy is lacking, I venture to give the history.

CASE 2.—The patient was a man of 65, of exemplary habits. His health had been good up to three years before, when he noticed at times a tight feeling in the precordia on walking. For the past three months typical anginal seizures often compelled him to stop after walking two or three blocks. Three days before he had had a moderately severe angina. Thirty-six hours before I first saw him, in the night he made a noise, awakening his wife. For a few seconds he was, perhaps, unconscious. He complained of unbearably severe pain in the upper stomach region; the pain did not radiate. He was nauseated and belched gas freely. His physician saw him inside of twenty minutes and gave sodium bicarbonate, which was vomited. The pain continuing, a hypodermic injection of morphin was necessary. The patient was pale, covered with cold sweat and had a small, rapid pulse. His appearance was that of collapse. His distress seemed to him largely abdominal.

When I saw him his color had returned and he was ruddy-cheeked. He complained of extreme weakness. His mind was clear. There was a little cyanosis, and respiration was somewhat labored. There were numerous râles in the chest. The pulse was 110 and small. The heart tones were faint; there was no murmur. The heart was a trifle enlarged, as it had been for some years. The area of cardiac flatness was decidedly small on account of overlying lung. The liver dulness was but a narrow band along the costal margin; the edge of the liver could be palpated. No spleen could be made out. The urine contained a distinct ring of albumin and a few granular and hyaline casts. There was a doubtful faint trace of bile. On digalen and nitroglycerin there seemed to be some improvement in the quality of the pulse.

At a second visit the condition was much the same. There had been a few periods of more marked oppression in breathing, with some increase in cyanosis and weakness of the pulse.

At a third visit, Oct. 19, 1910, it was learned that the patient had had a bad night, with severe attacks of dyspnea. The pulse had been but barely perceptible at the wrist and beat 120 to the minute. At 5 A.M. both the physician and the patient himself had felt that death was at hand. The patient had rallied, however, and when I saw him was conscious, with very feeble pulse of 110, and barely perceptible heart tones. He was extremely weak. Breathing was of the Cheyne-Stokes type. The patient seemingly dozed during apnea, yet answered questions. What I took to be a faint pericardial friction could be made out over the lower left sternal border. The patient said he was not in pain. He declared that he obtained relief

from swallowing orange juice, which he repeatedly sipped. He remained in this condition for sixteen hours longer. From the onset of his severe anginal attack to death was seven days.

The instructive case of Professor Panum is described by Fraentzel.[12] For a few weeks Panum had noticed dyspnea and a tight feeling on going up stairs. May 1, 1885, coming home in the wind, he stopped often, and on reaching home had a sudden, severe, tearing pain in the precordia, running out to the left arm and fingers. The pulse became rapid, small and irregular. The patient broke out into a profuse sweat. He was nauseated and induced vomiting by tickling his throat. The physical findings are not accurately known. The mind was clear to the last. Death occurred suddenly about fourteen hours after the onset of symptoms. At the necropsy both upper lobes of the lungs and the middle lobe were found emphysematous. The left coronary artery was atheromatous, narrowed, and a white soft thrombus was attached to the wall. The musculature of the left ventricle was degenerated and softened and had ruptured just to the left of the septum.

Engelhardt[13] describes the case of a man of 54 in whom, after a thrombosis of the left coronary artery with suddenly developing gastric and abdominal symptoms, there was an illness of eight days, with fever, meteorism, vomiting, oppression, and then in a tachycardial attack rupture of the anterior wall of the left ventricle, with hemopericardium. Death twelve hours after the rupture. The symptom-complex resembled the picture of the abdominal-pectoral vascular crises (Pal).

A study of cases of this type shows that nearly all are in men past the middle period of life. Previous attacks of angina have generally been experienced, though, as shown by my first case, the fatal thrombosis may bring on the first seizure. The seizure is described by patients who have had previous experience with angina as of unusual severity, and the pain persists much longer. In some instances there has been no definite radiation of the pain, as to the neck or left arm, though this may have been a feature of other anginal attacks, and the pain, as in these two cases, may be referred to the lower sternal region or definitely to the upper abdomen. Cases with little or no pain have been described. In Chiari's case pain is not referred to, the patient though with slow, irregular and weak pulse being out of bed. The obstruction of the right coronary was, as Chiari says, "so to speak, latent." Thorel also refers to a painless case. Some of Huchard's cases with obstruction did not show anginal pain. Nausea and vomiting, with belching of gas, are common. There may be tympany. Ashy countenance, cold sweat and feeble pulse complete the picture of collapse. The attention of the patient and the physician as well may, therefore, be strongly focused on the abdomen, and

[12]Fraentzel: Krankheiten des Herzens, Berlin, 1892. iii. 51.
[13]Engelhardt: Ein Fall von Herzruptur, Deutsch med. Wchnschr., xxxv, 1910, No 19, p 838.

some serious abdominal accident be regarded as the cause of the sudden pain, nausea, collapse. The cardiac origin may be the more easily overlooked when there has been no previous typical angina, and when, as may happen (Case 1), there is no arteriosclerosis manifested peripherally and no enlargement of the heart to be made out.

Cohnheim found that in dogs the pulse after obstruction was slow. This may be so in the thrombotic obstruction of disease in man. In Hammer's[14] case the pulse dropped from 80 to 8 per minute, the patient living thirty hours from the onset of the symptoms that marked the closure of the right coronary opening. A rapid pulse is frequently seen, however. The pulse may be irregular. A striking feature has been its weakness. In two patients I have seen a rapid, thready, almost imperceptible, radial pulse, of such a quality that if met with in pneumonia or typhoid fever, it would have warranted one in presaging death within a few minutes or hours. Yet one patient lived forty hours and another four or five days with a pulse of this quality. Blood-pressure is low. The heart tones have been feeble—in fact, often startlingly feeble. Feeble contraction of the weakened, anemic heart muscle accounts for the weak pulse and the weak tones. Still another reason for the faint tones is found in the acute emphysema—*Lungenschwellung* and *Lungenstarrheit* of von Basch—by which condition the heart sounds are obscured by overlapping air-containing lungs. This also makes it difficult to map out the outlines of the heart and, coupled with the feeble apex impulse, may make such an examination for the size of the heart very unsatisfactory.

Dyspnea and cyanosis have been variable, at times much less than one would expect from the character of the accident and the quality of the heart's action. Râles, dry and moist, have been present in many cases, in some, as in my first case, largely moist, diffuse, not very large. Here there was a moderate amount of thin, frothy, slightly blood-tinged fluid expectorated, as in edema of the lungs, which condition was found at the autopsy. I mention this because some, with Cohnheim, contended that the conditions for edema would not be produced by coronary obstruction, as both right and left heart ceased beating simultaneously. Others, e.g., Samuelson, Bettelheim and Michaelis, found edema. My case shows such edema. Possibly the right heart may have remained relatively stronger than the left after the accident, and so Welch's condition for edema has been presented.

The weakness of the heart and the low blood-pressure will account for the scanty urine and the trace of albumin. A palpable liver may likewise owe its enlargement to passive congestion.

Nearly always the mind is clear—at times unusually clear—until toward the last. Some patients seem conscious, as is so common in angina, that they are face to face with death, but in none that I have seen has

[14]Hammer: Wien. med. Wchnschr., 1878, No. 5.

there been uncontrollable fear or the restlessness of fright. The serious-ness of the accident seemed to be realized, but there was no panic. Per-haps the relief from the agony of the initial pain causes an unnatural mental calmness.

General weakness has been marked in some cases, in others not. One patient showed for more than a week an asthenia, comparable to that of the terminal stage of pernicious anemia or Addison's disease. He hesitated to move in bed for the further reason that even turning on the side caused him the sensation as though the heart were giving out. Even slight movement caused some pain. His case is representative of the type of status anginosus. Obrastzow regards this as the usual man-ifestation of coronary thrombosis. My experience shows that such ob-struction may be followed by a complete cessation of pain for hours, and even to the time of death. Some of these patients of the latter type will, if permitted, move freely or even get out of bed.

The occurrence of a serofibrinous exudate over the area of myocardial softening, with roughening of the pericardium, has been noted in several instances. This may explain a later precordial distress, as in Case 1. A fine pericardial friction, therefore, occurring several hours or a few days after the initial pain, may be confirmatory evidence of coronary obstruc-tion. Osler[15] concluded, in one of his cases of angina, that the attack was probably associated with acute infarct of the ventricle, "as a peri-cardial rub was detected the next day." Dock[9] recognized this peri-carditis *intra vitam* in one of his cases and found it post mortem over the softened area. In one of Leyden's cases in which the patient lived five days from the onset of symptoms of dizziness, faintness, small pulse, there was found myomalacia cordis, and especially at the apex, where a softened area reached the surface, there was pericarditis; cloudy fluid was in the pericardial sac. This was almost certainly a case of coronary obstruction, though the occluding lesion is not described. This peri-carditis is in keeping with some of the experimental work on lower ani-mals, e.g., that of Bickel, who in his dogs killed nineteen and seventy days after ligation found localized pericarditic adhesions over the area repre-senting the myocardial softening.

Death is the result in nearly all of these cases. Yet it may be delayed for many days. More than this, there is, as has been shown by reference to experimental work, no intrinsic reason why some patients with ob-struction of even large branches of the coronary artery may not re-cover. Experimental animals sometimes do. And as already said, mild cases must occur, and one cannot pretend to say where the dividing line should be drawn between the mild obstruction of a coronary branch, whose recovery means a few fibrous patches in the myocardium, and the more serious one that in a few days is to lead to rupture of the heart or is

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to produce an extensive weakened fibrous area that will ultimately yield in cardiac aneurysm. Death may then be caused by rupture, by sudden asystole, or by gradual giving out of the weakened heart muscle—by "ingravescent systole," as Balfour[16] styles it—a mode of death occupying from half an hour to a week, illustrated by one of his cases in which death occurred one week after the obstruction, which was found at post mortem. In one instance in which I believe the anginal seizure was thrombotic a dilatation of the heart, with orthopnea, dropsy, etc., followed the seizure. Death here was, as in cardiac failure, from other causes. Some of the dogs of Miller and Matthews died in this way several weeks after the ligation of the coronary. In cases in which the heart slowly wears out in the course of a few days, Cheyne-Stokes respiration, general asthenia, urinary scantiness, with mental apathy and exhaustion may be present.

Emphasis ought to be laid on the resemblance of some of these cases to surgical accidents. The sudden onset with pain over the lower sternal and epigastric region, the nausea and vomiting, the tympany, the feeble pulse, ashy color, cold sweat and other signs of collapse make one think of such conditions as gall-bladder disease, acute hemorrhagic pancreatitis, perforation of gastric or duodenal ulcer, hemorrhage into the adrenal capsule, etc. The dyspnea, hyperresonant thorax, obscured heart tones, may suggest pneumothorax or diaphragmatic hernia. In my first case, while the diagnosis made was that of cardiac accident, there were so many disquieting features that surgical counsel was called to make sure that some surgical accident, such as those enumerated, had not been overlooked. Details as to differential diagnosis need not be given. Where there is arteriosclerosis, enlarged heart, a history of previous angina, typical radiation of the pain to the neck and arm, the diagnosis will not be so difficult as where these suggestive aids are lacking. The bilateral character of the emphysema, the persistence of breath sounds, often with râles, the failure of the heart to be dislocated, will help exclude pneumothorax and diaphragmatic hernia. The absence of blood from the vomitus, the absence of peritonitic tenderness, a study of the temperature, the leukocytes, etc., will help in excluding subdiaphragmatic accidents.

Obrastzow[17] calls particular attention to this resemblance to surgical accidents which my own experience corroborates. Engelhardt's case also illustrates this point.

If these cases are recognized, the importance of absolute rest in bed for several days is clear. It would also seem to be far wiser to use digitalis, strophanthus or their congeners than to follow the routine practice of

[16]Balfour: Clinical Lectures on Diseases of the Heart, Edition 3, 1898, pp. 316 and 328.
[17]Obrastzow and Straschesko: Zur Kenntniss der Thrombose der Koronararterien des Herzens, Ztschr. f. klin. Med., 1910, lxxi, 116.

giving nitroglycerin or allied drugs. The hope for the damaged myocardium lies in the direction of securing a supply of blood through friendly neighboring vessels so as to restore so far as possible its functional integrity. Digitalis or strophanthus by increasing the force of the heart's beat, would tend to help in this direction more than the nitrites. The prejudice against digitalis in cases in which the myocardium is weak is only partially grounded in fact. Clinical experience shows this remedy of great value in angina, and especially in cases of angina with low blood pressure, and these obstructive cases come under this head. The timely use of this remedy may occasionally in such cases save life. Quick results should also be sought by using it hypodermically or intravenously. Other quickly acting heart remedies would also be of service.
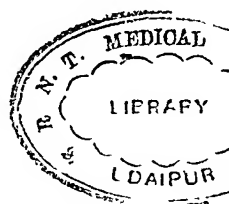
OLIVER WENDELL HOLMES

The Stethoscope Song
A Professional Ballad

By

OLIVER WENDELL HOLMES

There was a young man in Boston town,
He bought him a stethoscope nice and new,
All mounted and finished and polished down,
With an ivory cap and a stopper too.

It happened a spider within did crawl,
And spun him a web of ample size,
Wherein there chanced one day to fall
A couple of very imprudent flies.

The first was a bottle-fly, big and blue,
The second was smaller, and thin and long;
So there was a concert between the two,
Like an octave flute and a tavern gong.

Now being from Paris but recently,
This fine young man would show his skill;
And so they gave him, his hand to try,
A hospital patient extremely ill.

Some said his liver was short of bile,
And some that his heart was oversize,
While some kept arguing, all the while,
He was crammed with tubercles up to his eyes.

This fine young man then up stepped he,
And all the doctors made a pause;
Said he, The man must die, you see,
By the fifty-seventh of Louis's laws.

But since the case is a desperate one,
To explore his chest it may be well;
For if he should die and it were not done
You know the autopsy would not tell.

Then out his stethoscope he took,
And on it placed his curious ear;
Mon Dieu! said he with a knowing look,
Why, here is a sound that's mighty queer!

The *bourdonnement* is very clear,—
Amphoric buzzing, as I'm alive!
Five doctors took their turn to hear;
Amphoric buzzing, said all the five.

There's empyema beyond a doubt;
We'll plunge a trocar in his side.
The diagnosis was made out,—
They tapped the patient; so he died.

Now such as hate new-fashioned toys
Began to look extremely glum;
They said that rattles were made for boys,
And vowed that his buzzing was all a hum.

There was an old lady had long been sick,
And what was the matter none did know:
Her pulse was slow, though her tongue was quick;
To her this knowing youth must go.

So there the nice old lady sat,
With phials and boxes all in a row,
She asked the young doctor what he was at,
To thump her and tumble her ruffles so.

Now, when the stethoscope came out,
The flies began to buzz and whiz;
Oh, ho! the matter is clear, no doubt;
An aneurism there plainly is.

The *bruit de râpe* and the *bruit de scie*
And the *bruit de diable* are all combined;
How happy Bouillaud would be,
If he a case like this could find!

Now, when the neighboring doctors found
A case so rare had been described,
They every day her ribs did pound
In squads of twenty; so she died.

Then six young damsels, slight and frail,
Received this kind young doctor's care;
They all were getting slim and pale,
And short of breath in mounting stairs.

They all made rhymes with ''sighs'' and ''skies,''
And loathed their puddings and buttered rolls,
And dieted, much to their friends' surprise,
On pickles and pencils and chalk and coals.

So fast their little hearts did bound,
The frightened insects buzzed the more;
So over all their chests he found
The *râle sifflant* and the *râle sonore.*

He shook his head. There's grave disease,—
I greatly fear you all must die;
A slight post-mortem, if you would please,
Surviving friends would gratify.

The six young damsels wept aloud,
Which so prevailed on six young men
That each his honest love avowed.
Whereat they all got well again.

This poor young man was all aghast!
The price of stethoscopes came down;
And so he was reduced at last
To practice in a country town.

The doctors being very sore,
A stethoscope they did devise
That had a rammer to clear the bore,
With a knob at the end to kill the flies.

Now use your ears, all you that can,
But don't forget to mind your eyes,
Or you may be cheated, like this young man,
By a couple of silly, abnormal flies.
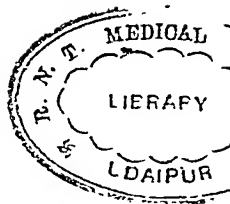
THE CORRELATION OF THESE CLASSICS WITH
OTHER CONTEMPORARY HISTORIC EVENTS

1788	Matthew Baillie	Description of congenital dextrocardia with complete situs transversus.	Scotland	The period of the French Revolution.
1791	John Hunter	Classic description of his anginal attacks.	England	Treaty with the United States of America (Jay's treaty).
1806	Jean Nicolas Corvisart	Description of the signs of contraction of the orifices of the heart, etc.	France	France, under Napoleon, at war with Prussia and Russia.
1812	William Charles Wells	The earliest account relating rheumatic fever with heart disease.	United States and England	The War of 1812 between the United States and Great Britain.
1818	John Cheyne	Description of that periodic type of breathing later to be known as Cheyne-Stokes respiration.	Scotland	The reign of George III, three years after the battle of Waterloo.
1819	René T. H. Laënnec	The introduction of the stethoscope and auscultation.	France	Two years before the death of Napoleon I.
1825	Caleb Parry	Description of the circulatory phenomena of exophthalmic goiter.	England	Financial panic and failure of banks.
1827	Robert Adams	Description of heart block.	Ireland	Battle of Navarino, destruction of the Turkish fleet.
1831	James Hope	Description of cardiac asthma, stenosis of the pulmonary valves, and cardiac neurosis.	England	British Association for the Advancement of Science held its first meeting.
1832	Dominic John Corrigan	Description of the pulse in aortic insufficiency.	Ireland	Irish reform act passed.
1835	Jean Baptiste Bouillaud	The pathology of endocarditis.	France	Death of Dupuytren, the famous French surgeon.
1846	William Stokes	Description of heart block.	Ireland	Failure of the potato crop in Ireland resulting in famine.
1852	William Senhouse Kirkes	Discussion of emboli from intracardiac ganglia.	England	Disraeli was Chancellor of the Exchequer.
1854	William Stokes	Description of that periodic type of breathing later to be known as Cheyne-Stokes respiration.	Ireland	Reciprocity treaty between the United States and Great Britain.

THE CORRELATION OF THESE CLASSICS WITH OTHER CONTEMPORARY HISTORIC EVENTS—CONT'D

YEAR	AUTHOR	CONTRIBUTION INCLUDED IN THIS VOLUME	COUNTRY	CONTEMPORARY HISTORIC EVENT
1861	Paul Louis Duroziez	Description of the femoral murmurs in aortic insufficiency.	France	Mexican Expedition.
1862	Austin Flint	Description of the murmur later to be known as the Austin Flint murmur.	United States	One year following Lincoln's inauguration as President. Civil War one year old.
1867	Pierre Carl E. Potain	Description of the pulsations in the jugular veins.	France	The Luxembourg question.
1867	Thomas Lauder Brunton	Introduction of amyl nitrite in the treatment of angina pectoris.	England	The formation of the Dominion of Canada.
1868	Heinrich Quincke	Demonstration of the capillary and venous pulse.	Germany	A year before the formal opening of the Suez Canal.
1870	Samuel Wilks	Description of bacterial endocarditis.	England	Death of Charles Dickens.
1872	Ludwig Traube	Demonstration of pulsus alternans.	Germany	A year after the close of the Franco-Prussian War.
1876	William Richard Gowers	Description of the ocular fundi in hypertension.	England	Victoria proclaimed Empress of India.
1877	Julius Cohnheim	Description of paradoxical embolism.	Germany	Turco-Russian War.
1879	Henri Roger	Description of the murmur of patency of the interventricular septum.	France	Jules Grévy President of the Third Republic of France.
1879	William Murrell	The introduction of nitroglycerin in the treatment of angina pectoris.	England	Strike of London engineers against reduction of wages.
1885	Pierre Carl E. Potain	Discussion of gallop rhythm.	France	Death of Victor Hugo.
1887	Augustus D. Waller	Demonstration of a method of leading the action currents of the heart from the surface of the heart.	England	Triple Alliance renewed.

Year	Name	Contribution	Country	World Events
1887	John A. MacWilliam	Experimental production of various cardiac arrhythmias.	England	Nordenfelt's boat submarine successfully tried at Southampton.
1888	Graham Steell	Description of the murmur of high pressure in the pulmonary artery, later to be known as the Graham Steell murmur.	England	Death of Matthew Arnold.
1888	Etienne Louis A. Fallot	Description of the "maladie bleue," later to be known as the tetralogy of Fallot.	France	67,000,000 francs voted for the defense of Brest, Toulon, and Cherbourg.
1893	Wilhelm His, Jr.	Demonstration of the auriculoventricular bundle (bundle of His).	Germany	Tariff controversy between Germany and Russia in progress.
1896	Francis H. Williams	Early study of fluoroscopy of the heart and aorta.	United States	Election of President McKinley.
1897	William H. Broadbent	Classic description of adherent pericarditis.	England	Workmen's Compensation Act approved.
1903	Willem Einthoven	Introduction of the string galvanometer (electrocardiograph).	Holland	Gift of $1,500,000 by Andrew Carnegie for a temple of peace for the permanent court of arbitration at The Hague, reported.
1904	Ludwig Aschoff	Classic description of rheumatic myocarditis (Aschoff nodule).	Germany	Arbitration treaty between Great Britain and Germany.
1907	Arthur Keith and Martin Flack	Demonstration of the sinoauricular node and its function as the pacemaker of the heart.	England	Death of Sir Michael Foster, famed physiologist.
1908	James Mackenzie	Description of auricular fibrillation.	Scotland	Wireless telegraph extended between Montreal and London.
1909	William Osler	Description of the skin nodules in subacute bacterial endocarditis, later to be known as Osler nodes.	Canada, United States, England	Peary's expedition to the North Pole.
1912	James B. Herrick	Classic description of coronary thrombosis.	United States	New Mexico and Arizona admitted to the Union.

REFERENCES

William Harvey

Garrison, F. H.: An introduction to the history of medicine, with medical chronology, suggestions for study and bibliographic data, ed. 4, Philadelphia, 1929, W. B. Saunders Company, 996 pp.

Power, D'Arcy: William Harvey, London, 1897, T. F. Unwin, 283 pp.

Smith, Preserved: A history of modern culture, New York, 1930, Henry Holt & Company, Inc., vol. 1, 672 pp.; vol. 2, 703 pp.

The works of William Harvey, M.D., translated from the Latin, with a life of the author, by Robert Willis, London, 1847, The Sydenham Society, 1906, 624 pp.

Pierre Gassendi

Tallmadge, G. K.: Pierre Gassendi and the "Elegans de septo cordis pervio observatio," Bull. Hist. Med. 7: 429-457, 1939.

Marcello Malpighi

Foster, Michael: Lectures on the history of physiology during the sixteenth, seventeenth and eighteenth centuries, Cambridge, 1901, University Press, 310 pp.

Garrison, F. H.: op. cit.

Niels Stensen

Lutz, F. J.: Nicholas Steno, M. Libr. & Hist. J. 2: 166-182, 1904.

Miller, W. S.: Niels Stensen, Bull. Johns Hopkins Hosp. 25: 44-51, 1914.

William Cowper

Archaeologica medica: XLI. William Cowper, the anatomist, Brit. M. J. 1: 160-161, 1898.

Beeckman, Fenwick: Bidloo and Cowper, anatomists, Ann. M. Hist. n.s. 7: 113-129, 1935.

McIntosh, W. A.: Cowper—the anatomist, Canad. M. A. J. 10: 938-945, 1920.

Antony van Leeuwenhoek

DeKruif, P. H.: Microbe hunters, New York, 1926, Harcourt, Brace & Company, Inc., 363 pp.

Dobell, Clifford: A protozoological bicentenary; Antony van Leeuwenhoek (1632-1723) and Louis Joblot (1645-1723), Parasitology 15: 308-319, 1923.

De Graaf, Regnerus: Some observations made by M. Leeuwenhoek in Holland, Phil. Tr. Roy. Soc. London 2: 66-67, 1672-1683. (From the abridged edition published in 1809.)

Nuttall, G. H. F.: Notes bearing on Leeuwenhoek, Redi, Linnaeus, De Geer, Rudolphi, Müller, Leidy, Owen, Jenner, Kircher and Boyle, whose portraits have appeared in Parasitology, vol. XIII (portrait-plates I-XI), Parasitology 13: 398-400, 1921.

Plimmer, H. G.: The president's address: "Bedellus immortalis," J. Roy. Micr. Soc. 33: 121-135, 1913.

Stephen Hales

Archaeologica medica: XXXVI. Stephen Hales, a pioneer in modern physiology, Brit. M. J. 2: 1191, 1897.

Burget, G. E.: Stephen Hales (1677-1761), Ann. M. Hist. 7: 109-116, 1925.

Dawson, P. M.: The biography of Stephen Hales, D.D., F.R.S., Bull. Johns Hopkins Hosp. 15: 185-192, 1904.

Dawson, P. M.: Stephen Hales, the physiologist, Bull. Johns Hopkins Hosp. 15: 232-237, 1904.

Jean Baptiste De Sénac

Degris, Georges: Étude sur Sénac, premier médecin de Louis XV (1693-1770), Paris, 1901, L. Boyer, 78 pp.

Long, E. R.: A history of pathology, Baltimore, 1928, Williams and Wilkins Company, 291 pp.

Renouard, Pierre-Victor: History of medicine, from its origin to the nineteenth century, with an appendix, containing a philosophical and historical review of medicine to the present time, translated from the French by C. G. Comegys, Cincinnati, 1856, Moore, Wilstach, Keys and Co., 719 pp.

Albrecht von Haller

Fulton, J. F.: Annual oration of the Boston Medical Library: Haller and the humanization of bibliography, New England J. Med. 206: 323-328, 1932.

Garrison, F. H.: op. cit., p. 317-319.

Hemmeter, J. C.: Albrecht von Haller; scientific, literary, and poetical activity, Bull. Johns Hopkins Hosp. 19: 65-74, 1908.

Heroes of medicine: Albrecht von Haller, Practitioner 57: 623-627, 1896.

Pettigrew, T. J.: ''Albert de Haller, M.D., F.R.S,'' in Medical portrait gallery; biographical memoirs of the most celebrated physicians, surgeons, etc., who have contributed to the advancement of medical science, London, 1840, Fisher, Son & Co., vol. 1, 10 pp.

John Baptist Morgagni

Adams, E. W.: Founders of modern medicine: II. Giovanni Battista Morgagni (A.D. 1682-1771), M. Libr. & Hist. J. 1: 270-277, 1903.

Long, E. R.: op. cit.

Nicholls, A. G.: Morgagni, the father of modern pathology; an appreciation and a comparison, Montreal M. J. 32: 40-51, 1903.

Pettigrew, T. J.: ''John Baptist Morgagni, M.D., F.R.S., etc,'' op. cit., vol. 1, 4 pp.

Leopold Auenbrugger

Dickens, Charles: The mystery of Edwin Drood and Master Humphrey's clock, New York, 1901, Charles Scribner's Sons, p. 36.

Nova et vetera: Leopold Auenbrugger; the discovery of percussion, Brit. M. J. 1: 1191-1192, 1909.

Otis, E. O.: Auenbrugger and Laënnec, the discoverers of percussion and auscultation, Boston M. & S. J. 139: 281-285; 305-308, 1898.

William Heberden

Davidson, P. B.: William Heberden, M.D., F.R.S., Ann. M. Hist. 4: 336-346, 1922.

Garrison, F. H.: op. cit.

Heberden, William: An introduction to the study of physic [now for the first time published]. A prefactory essay by LeRoy Crummer, with a reprint of Heberden's ''Some account of a disorder of the breast,'' New York, 1929, Paul B. Hoeber, Inc., 159 pp.

Pettigrew, T. J.: ''William Heberden, M.D., F.R.S., etc.,'' op. cit., London, 1840, Whittaker & Co., vol. 3, 18 pp.

William Withering

Cushny, A. R.: William Withering, M.D., F.R.S., Proc. Roy. Soc. Med. (Sect. Hist. Med.) 8: 85-94, 1915.

Garrison, F. H.: op. cit.

Kolipinski, Louis: William Withering and his book on the foxglove, M. Rec. 86: 8-14, 1914.

Roddis, L. H.: William Withering and introduction of digitalis into medical practice, Ann. M. Hist. n.s. 8: 93-112; 185-201, 1936.

Shryock, R. H.: The development of modern medicine; an interpretation of the social and scientific factors involved, Philadelphia, 1936, University of Pennsylvania Press, pp. 1-2.

Matthew Baillie

Heroes of medicine: Baillie, Matthew. An autobiography, entitled "A short memoir of my life, with a view of furnishing authentic materials," edited by J. B. Bailey, Practitioner 57: 51-65, 1896.

Macmichael, William: The gold-headed cane, with an introduction by Sir William Osler and a preface by Francis R. Packard, New York, 1915, Paul B. Hoeber, Inc., pp. 225-253.

Pettigrew, T. J.: "Matthew Baillie, M.D., F.R.S.," op. cit., vol. 2, 10 pp.

John Hunter

Garrison, F. H.: op. cit.

Ottley, Drewry: "The life of John Hunter, F.R.S.," in The works of John Hunter, with notes, edited by J. F. Palmer, London, 1835, Longman, etc., vol. 1, pp. 1-198.

Parry, T. W.: Immortal names and other poems, London, 1937, Mitre Press, 122 pp.

Jean Nicolas Corvisart

Beeson, B. B.: Corvisart, his life and works, Ann. M. Hist. n.s. 2: 297-307, 1930.

William Charles Wells

Bartlett, Elisha: A brief sketch of the life, character, and writings of William Charles Wells, M.D., F.R.S. An address delivered before the Louisville Medical Society, December 7th, 1849, Louisville, Kentucky, 1849, Prentice & Weissinger, 32 pp.

Beck, J. B.: An historical sketch of the state of medicine in the American colonies, from their first settlement to the period of the revolution, ed. 2, Albany, New York, 1850, Charles van Benthuysen, Printer, 63 pp.

James, R. R.: William Charles Wells, 1757-1817, Brit. J. Ophth. 12: 561-569, 1928.

Keil, H.: Dr. William Charles Wells and his contribution to the study of rheumatic fever, Bull. Inst. Hist. Med. 4: 789-816, 1936.

Pleadwell, F. L.: "That remarkable philosopher and physician, Wells of Charleston," the first exponent of the theory of natural selection, discoverer of the nature of dew, and physician, Ann. M. Hist. n.s. 6: 128-142, 1934.

Roberts, S. R.: William Charles Wells of Charleston and London, Boston M. & S. J. 193: 1140-1145, 1925.

John Cheyne

Pettigrew, T. J.: "John Cheyne, M.D., F.R.S.E., M.R.I.A.; physician general to the forces in Ireland," op. cit., vol. 3, 20 pp.

René T. H. Laënnec

Garrison, F. H.: Laënnec (editorial), Bull. New York Acad. Med. s.2. 2: 389-393, 1926.

Medical Intelligence (news note): Monument to Laënnec, Boston M. & S. J. 76: 382, 1867.

Robinson, Victor: René Théophile Hyacinthe Laënnec (1781-1826), M. Life 28: 456-469, 1921.

Webb, G. B.: René Théophile Hyacinthe Laënnec; a memoir, New York, 1928, Paul B. Hoeber, Inc., 146 pp.

Caleb Parry

Rolleston, Sir Humphry: Caleb Hillier Parry, M.D., F.R.S., Ann. M. Hist. 7: 205-215, 1925.

Robert Adams

Major, R. H.: Classic descriptions of disease, with biographical sketches of the authors, Springfield, Illinois, 1932, Charles C. Thomas, 630 pp.

Obituary: Robert Adams, M.A., M.D., Lancet 1: 145, 1875.

James Hope

Complete works of George Gordon Byron, Boston, Houghton, Mifflin Company. p. 99.

Flaxman, N.: The hope of cardiology; James Hope (1801-1841), Bull. Inst. Hist. Med. 6: 1-21, 1938.

Hope, Mrs. (Anne): Memoir of the late James Hope, M D., physician to St. George's Hospital, &.&., to which are added, remarks on classical education, by Dr. Hope; and letters from a senior to a junior physician, by Dr Burder; the whole edited by Klein Grant, M.D., etc., ed. 3, London, 1844, J. Hatchard and Son, 365 pp.

Keats, John, and Shelley, P. B.: Complete poetical works, with the explanatory note of Shelley's poems by Mrs. Shelley, New York, The Modern Library, p. 366.

Dominic John Corrigan

Dock, George: I. Dominic John Corrigan; his place in the development of our knowledge of cardiac disease: II. The water-hammer pulse, Ann. M. Hist. n.s. 6: 381-395, 1934.

Obituary: Sir Dominic John Corrigan, Bart., M.D., Brit. M. J. 1: 227-228, 1880.

Rolleston, Sir Humphry: Sir Dominic Corrigan, Bart , M.D , Irish J M. Sc., 1932, June, pp. 261-266.

Williamson, R. T.: Sir Dominic Corrigan, Ann. M. Hist. 7. 354 361, 1925.

Jean Baptiste Bouillaud

Lereboullet, P.: J. Bouillaud (1796-1881), Paris méd. 16: 177 183, 1914

Rolleston, J. D.: Jean Baptiste Bouillaud (1796-1881). A pioneer in cardiology and neurology, Proc. Roy. Soc. Med. (Sect. Hist. Med.) pt. 2 24: 1253 1262, 1931.

William Stokes

Stokes, William: William Stokes, his life and work (1804-1878) by his son, William Stokes, London, 1898, T. F. Unwin, 256 pp.

William Senhouse Kirkes

Obituary: William Senhouse Kirkes, M.D., F.R.C.P., Lancet 2: 674, 1864.

Power, D'Arcy: William Senhouse Kirkes, M.D., St. Barth. Hosp. J. 18: 166-168, 1910-1911.

Paul Louis Duroziez

Helfenbein, Feiga: Duroziez et son oeuvre, contribution à l'histoire de la cardiologie, Paris, 1922, no. 430, 36 pp. Thesis.

Austin Flint

Carpenter, W. M.: Memoir of Austin Flint, M.D., L.L.D., Tr. M. Soc. New York 81: 556-565, 1887.

Landis, H. R. M.: Austin Flint: his contributions to the art of physical diagnosis and the study of tuberculosis, Bull. Johns Hopkins Hosp. 23: 182-186, 1912.

Necrology: Austin Flint, Sr., M.D., LL.D., J. A. M. A. 6: 361-364, 1886.

Walsh, J. J.: History of medicine in New York, three centuries of medical progress, New York, 1919, National Americana Society, Inc., vol. 4, pp. 14-15.

Pierre Carl E. Potain

Vaquez, H.: Pierre-Carl Potain (1825-1901): éloge prononcé à l'académie de médecine dans la séance annuelle du 13 Decembre 1927, Bull. Acad. de méd. Paris s.3. 98: 569-587, 1927.

Thomas Lauder Brunton

Garrison, F. H.: Sir Thomas Lauder Brunton, M.D. (1844-1916), an apostle of preparedness, Mil. Surgeon 40: 369-377, 1917.

MacDougall, J. A.: Obituary: Sir Lauder Brunton, Bart., M.D., C.M., D.Sc. (Edin.), R.U.I. (Hon.), LL.D. (Edin. and Aberd.), F.R.C.P., F.R.S., Consulting Physician to St. Bartholomew's Hospital, Edinburgh M. J. n.s. 17: 345-349, 1916.

Obituary: Sir Thomas Lauder Brunton, Bt., M.D., F.R.S.; consulting physician to St. Bartholomew's Hospital, Brit. M. J. 2: 440-442, 1916.

Obituary: Sir T. Lauder Brunton, Bart., M.D., C.M., D.Sc., Edin., M.D.R.U.I. (Hon.), LL.D. Edin. and Aberd. (Hon.), F.R.C.P. Lond., F.R.S.; consulting physician to St. Bartholomew's Hospital, Lancet 2: 572-575, 1916.

Power, D'Arcy: In memoriam: Sir Thomas Lauder Brunton, F.R.S., 1844-1916, St. Barth. Hosp. Rep. 52: 1-9, 1916.

Heinrich Quincke

Annotations: Heinrich Quincke, Lancet 1: 1259, 1922.

von Bergmann, G.: Heinrich Quincke. Gedenkrede bei einer Trauerfeier der medizinischen Fakultät und des Ärztlichen Vereins zu Frankfurt am Main, Ztschr. f. klin. Med. 96: 1-21, 1923.

Hochhaus, H.: Heinrich Quincke zum 70. Geburtstag, Deutsche med. Wchnschr. 2: 1605-1607, 1912.

Hoppe-Seyler, G.: Heinrich Quincke, München. med. Wchnschr. 69: 1014-1016, 1922.

Külbs: Heinrich Quincke, Deutsches Arch. f. klin. Med. 140: i-iv, 1922.

Samuel Wilks

White, W. Hale: Samuel Wilks, Guy's Hosp. Rep. s.3. 52: 1-39, 1913.

Ludwig Traube

Boruttau, H.: Ludwig Traube und die Geschichte der experimentellen Pathologie in Deutschland (kritische Bemerkungen), Ztschr. f. exper. Path. u. Therap. 20: 144-148, 1919.

Correspondence: The death of Louis Traube, M. Rec. 11: 323-324, 1876.

Haberling, W.: Zum hundertsten Geburtstag Ludwig Traubes, Deutsche med. Wchnschr. 1: 21-23, 1918.

von Leyden, E.: Gedächtnissrede auf Ludwig Traube, Charité-Ann. 2: 767-800, 1877.

Morrison, Hyman: Ludwig Traube, Boston M. & S. J. 196: 1097-1101, 1927.

Virchow, Rud.: Zur Erinnerung an L. Traube, Berl. klin. Wchnschr. 13: 209-210, 1876.

William Richard Gowers

Langdon, F. W.: Sir W. R. Gowers, M.D., F.R.C.P., F.R.S.; an appreciation, Lancet-Clinic 114: 142-144, 1915.

Obituary: Sir William Gowers, M.D., F.R.C.P., F.R.S.; consulting physician to University College Hospital and to the National Hospital for the Paralysed and Epileptic, London, Brit. M. J. 1: 828-830, 1915.

Obituary: Sir William Richard Gowers, M.D. Lond. and Dub., F.R.C.P. Lond., LL.D. Edin., F.R.S.; consulting physician to University College Hospital, London, and to the National Hospital for the Paralysed and Epileptic, Queen-Square, W.C., Lancet 1: 1055-1056, 1915.

Obituary: Sir William Richard Gowers, M.D., F.R.C.P., F.R.S., Boston M. & S. J. 172: 877-878, 1915.

Julius Friedrich Cohnheim

McKee, A. B.: Memoir in his translation of Cohnheim's Lectures on general pathology; a handbook for practitioners and students, London, 1889, The New Sydenham Society, vol. 1, pp. ix-xv.

Rohrer, C. W. G.: Researches in cancer: Part I, Baltimore, 1934, The Brentwood Printing Company, pp. 49-126.

Henri Roger

Lereboullet, P.: Henri Roger (1809-1891), Paris méd. 14: 25, 1913.

Obituary: Henri Roger, Mém. et bull. soc. de méd. et chir. de Bordeaux (1892), pp. 5-6, 1893.

William Murrell

Obituary: William Murrell, M.D., Brux., F.R.C.P. Lond.; senior physician to, and lecturer on medicine at, Westminster Hospital, Lancet 2: 124, 1912.

Spencer, W. G.: In memoriam: William Murrell, M.D., F.R.C.P., Westminster Hosp. Rep. 18: 11-15, 1911-1912 (published in 1913).

Augustus Désiré Waller

Obituary: Augustus D. Waller, M.D., LL.D., F.R.S.; director of the physiological laboratory of the University of London, Brit. M. J. 1: 458-459, 1922.

Obituary: Augustus Désiré Waller, M.D., C.M. Aberd., D.Sc. Perth, W. A., Hon. LL.D. Edin., Hon. M.D. Geneva, F.R.S.; director of the physiological laboratory, University of London, Lancet 1: 555, 1922.

John Alexander MacWilliam

Cruickshank, E. W. H.: Prof. J. A. MacWilliam, F.R.S., Nature (London) 139: 224-225, 1937.

Obituary: John Alexander MacWilliam, M.D., LL.D. Aberd., F.R.S., Lancet 1: 236-237, 1937.

Louis A. Fallot

Olmer, D.: ''Étienne-Louis Arthur Fallot'' in Leçon d'ouverture de la chaire de clinique medicale, Marseille méd. 66: 741-759, 1929.

Wilhelm His

Blech, G. M.: Obituary: Dr. Wilhelm His, Mil. Surgeon 76: 99, 1935.

His, Wilhelm: Zur Geschichte des Atrioventrikularbündels nebst Bemerkungen über die embryonale Herztätigkeit, Klin Wchnschr. 12: 569-574, 1933.

In Memoriam Wilhelm His, Klin. Wchnschr. 13: 1689-1692, 1934.

von Krehl, L.: Wilhelm His zum 29. Dezember 1933, München. med. Wchnschr. 2: 2044-2045, 1933.

Obituary: Wilhelm His, Jr., Lancet 2: 1200, 1934.

Staehelin, R.: Wilhelm His zum siebzigsten Geburtstage, Klin. Wchnschr. 12: 1995-1996, 1933.

Schilling, V.: Wilhelm His (geboren 29.12.63; gestorben 10.10.34), Deutsches Arch. f. klin. Med. 177: i-viii, 1934.

Francis Henry Williams

Brown, Percy: American martyrs to science through the roentgen rays, Springfield, Illinois, 1936, Charles C. Thomas, 276 pp.

Brown, Percy: Francis Henry Williams, 1852-1936, Am. J. Roentgenol. 36: 106-110, 1936.

William H. Broadbent

Leaders in modern medicine: VII. Sir William H. Broadbent, Bart., Practitioner 81: 329-335, 1908.

Obituary: Sir William Henry Broadbent, Bart., K.C.V.O., M.D. Lond., F.R.C.P. Lond., LL.D. Edin. & St. And. & Toronto, D.Sc. Leeds, F.R.S., Commander of the Legion of Honour; physician in ordinary to H.M. the King and H.R.H. the Prince of Wales; consulting physician to St. Mary's Hospital, the London Fever Hospital, and the New Hospital for Women, Lancet 2: 126-130, 1907.

Obituary: Sir William Henry Broadbent, Bart., K.C.V.O., M.D. Lond., F.R.C.P., F.R.S., Hon. LL.D. Edin., St. And., and Toronto, Hon. D.Sc. Leeds; physician in ordinary to the King and to the Prince of Wales; consulting physician to St. Mary's Hospital, Brit. M. J. 2: 177-181, 1907.

Willem Einthoven

Kristenson, A.: Professor Willem Einthoven, Upsala läkaref. förh. 33: xi-xvi, 1928.

Leaman, W. G., Jr.: The history of electrocardiography, Ann. M. Hist. n.s. 8: 113-117, 1936.

Lewis, Thomas: Willem Einthoven, professor of physiology, University of Leyden, Heart 14: v-viii, 1928.
Obituary: Willem Einthoven (1860-1927), Dr. Med., Boston M. & S. J. 197: 687-688, 1927.
Wenckebach: W. Einthoven, Deutsche med. Wchnschr. 2: 2176, 1927.
Winterberg, H.: W. Einthoven, Wien. klin. Wchnschr. 40: 1460, 1927.

Ludwig Aschoff

Büchner, Franz: Ludwig Aschoff zum siebzigsten Geburtstag, Klin. Wchnschr. 15: 1-2, 1936.
Dietrich, A.: Ludwig Aschoff zum 70. Geburtstag, München. med. Wchnschr. 1: 61-62, 1936.
Ernst, Paul: Ludwig Aschoff zum 60. Geburtstage (10. Januar 1926), Med. Klin. 1: 79-80, 1926.
Koch, Walter: Ludwig Aschoff (zum 10. Januar 1926), München. med. Wchnschr. 1: 74-75, 1926.
The Doctor's Page: Ludwig Aschoff, J. Organotherapy 18: 203-204, 1934.
Wätjen, J.: Ludwig Aschoff zum 70. Geburtstag, Deutsche med. Wchnschr. 1: 79-80, 1936.

Arthur Keith

Dedicatory note to Sir Arthur Keith—a beloved master of British anatomy, J. Anat. 69: 1, 1934.
Keith, Sir Arthur: Personal communication to the authors.
"Keith, Sir Arthur," in Who's who, London, 1938, A. & C. Black, Ltd., vol. 19, p. 1834.
"Keith, Sir Arthur," in Medical directory, London, 1939, J. & A. Churchill, Ltd., vol. 95, p. 840.

Martin Flack

Kean, J. R.: Obituary: Martin Black, Mil. Surgeon 69: 570, 1931.
Obituary: Martin William Flack, C.B.E., M.B. Oxf.; director, Medical Research Department, R.A.F., Lancet 2: 430, 1931.
Obituary: Group-Captain Martin Flack, C.B.E., M.A., Oxon., M.B., B.Ch. Oxon., Brit. M. J. 2: 402-404, 1931.
Obituary: The late Group-Captain Martin Flack, Lancet 2: 510, 1931.

James Mackenzie

Wilson, R. M.: The beloved physician, Sir James Mackenzie, New York, 1926, The Macmillan Company, 316 pp.

William Osler

Memorial number of Canadian Medical Association Journal: Sir William Osler, Canad. M. A. J. (July) 1920, 123 pp.
Cushing, Harvey: The life of Sir William Osler, Oxford, 1925, The Clarendon Press, 2 vol.

James B. Herrick

Herrick, J. B.: N. S. Davis, Bull. Soc. M. Hist. of Chicago 4: 403-413, 1935.
Holmes, W. H.: James Bryan Herrick; an appreciation, privately printed in November, 1935, for The Central Interurban Clinical Club, 45 pp.

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(Names in boldface are those of authors whose contributions appear; numbers in boldface refer to biographic data.)

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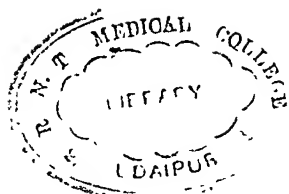
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